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The studies which have been conducted in past decades regarding lipid lipoprotein profile had been largely in light of atherosclerosis (AS), a type of arteriosclerosis (Robins and Cotrans: Pathologic basis of disease, 1984), an entity having potentiality of causing various diseases. Although any artery might be affected, the aorta, the coronaries and the cerebral systems are the prime target and thus myocardial infarcts and cerebral infarcts (strokes) are two major consequences therefore listing it as major cause of morbidity and mortality.

This AS is the outcome of interactions of many factors viz. obesity, diabetes mellitus, stress, family history of IHD, sedentary habits, hypercholesterolemia, age, smoking, hypertension and sex. Although diet is not considered a primary risk factor for cardiovascular disease but it does contribute to propagation of many other risk factors including high lipid lipoprotein levels, obesity etc. Thus diet has vital play since modifications in diet has led to progression or regression of AS lesions in experimental roles (Jackson et al, 1980). Whatever may be the interplay among risk factors, abnormal lipid lipoprotein profile may be common to most of these. Individual responses to high cholesterol fat diet varies enormously but remains constant for an individual over a long period of time (Kingsbury, 1960; Beynen et al,
1985). The study of Grunda and Vega (1982) also arrived at same conclusion and on its basis they introduced the term hypo and hyper responders, depending upon the response.

AS, disease of large and medium sized muscular arteries, has a basic lesion - the atheroma or fibro-fatty plaque and a covering fibrous cap (Robins and Cotrans, 1984). It may thus be very essential to understand the definition of AS, which has been defined as (WHO, 1969) a variable combination of changes of the intima consisting of the accumulation of lipids, complex carbohydrates, blood and its constituents, fibrous tissue, and calcium deposits in combined form. Recently, tissue culture techniques have demonstrated that LDL is rapidly taken up by arterial smooth muscles and endothelial cells. This can probably be explained by the identification of LDL receptors by Joseph Goldstein due to which LDL may rapidly be taken up. Thus LDL is the main culprit in atherogenesis.

Perhaps, overemphasis has been give to the importance of basal fasting cholesterol level as an individual risk for coronary artery disease. This is inferred from the study (Gregory et al, 1983) that more than 40% of young patients of CAD do not reveal raised fasting cholesterol levels and yet have atherogenous vascular involvement. Therefore fasting lipid lipoprotein profile does not truly reflect true risk of an
individual. In that case feeding induced changes in lipid lipoprotein profile may be more helpful in screening out susceptible individuals. Moreover, reproducibility of changes in lipid lipoprotein profile by similar protocols in same individuals after a period of few years may be a better marker for screening susceptible ones.

To explain the above, it has been postulated that atherogenesis may be a post-prandial phenomenon (Zilversmit, 1973). Transient post-prandial rise of various lipoproteins fractions (Beta VLDL, chylomicrons etc) causes repeated cholesterol deposition in cells in arterial walls over many years while fasting values may remain well within normal range over this duration. To have a deeper understanding it may be essential to know that cholesterol of dietary origin is transformed into various lipoproteins classes and thus contributes to elevation of total plasma cholesterol. More so, the amount and quantity of fat in diet have well documented effect upon plasma lipid concentration i.e. dietary saturated fatty acids have a hypercholesterolemic effect and increase LDL, monosaturated fatty acids have no other effect on plasma lipids while polyunsaturated fatty acids in general decreases plasma cholesterol and LDL concentration (Ahrens et al, 1967). However, LDL has positive correlation between it and risk of IHD while inverse relation holds true for HDL (Heirs et al, 1980).
In the Framingham study the cholesterol levels in male below age of 40 years was closely related to future development of IHD while this relation was less pronounced in elderly subjects. For both sexes, the relative incidence of IHD in subjects between 30-49 years age with cholesterol levels 7260 mg% was three to five times higher than those having value less than 220 mg%.

The process of AS begins in the childhood 1-10 years of age (WHO 1965-77) and Newmann et al (1986). Thus risk factors may well be identified in children but no prospective studies show relationship between presence of these risk factors in children to development of premature disease in later life. However, one can reasonably assume that if intervention are initiated early than there are greater likelihood of preventing cardiovascular diseases.

There are striking sex difference of disease prevalence (Strong et al, 1979) being higher in males and more so along with familial predisposition. In women low incidence of CAD before menopause has been attributed to oestrogen due to which they have lower lipid lipoprotein profile than in males. Many studies support this point due to fact that menopause is associated with increased risk of IHD and the incidence is comparable to male of similar age groups (Bangtson

Previous years studies in our department over serum lipid lipoprotein profile over nearly all age groups of subjects of both sexes, revealed that response to high cholesterol fat diet (HCFD) is largely variable. Young male subjects showed a greater percentage increase in the serum total cholesterol (STC) after 7 days of HCFD and this raised STC reverts back to normal after seven more days of withdrawal of HCFD. The rise of STC was attributed to HDL, while in young females HDL fraction increased significantly after 7 days of HCFD and further increased after withdrawal for 7 more days. While STG showed decrease in first 7 days of HCFD and later reverts back to normal after withdrawal of HCFD. Conversely in middle aged males/ menopausal females rise in STC after 7 days of HCFD was much less marked (Arora et al, 1984-1989) and was due to raised LDL fraction.

In order to have a better understanding and viewing for a better marker to screen susceptible ones reproducibility modes may be chosen. In this study a large number of identifiable normal healthy subjects who were to be directly and strictly scrutinized for
HCFD responses and who had earlier undergone study for lipid lipoprotein profile, were re-studied with same protocol after a gap of 3-7 years. They were given HCFD only at breakfast and then the serum lipid lipoprotein was studied with following aims:

1. To assess the changes in fasting basal lipid lipoprotein profile and those after HCFD.

2. Whether these changes, which were reproducible after a period of 3-7 years for healthy normal individuals both male and female showed any variation of patterns or not.

3. To study the possible association of changes seen with risk factor and correlate them quantitatively and qualitatively with possible development of atherosclerotic associated diseases, especially C.A.D.