DISCUSSION
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A single high cholesterol diet induced change in various subfractions of lipid lipoproteins and its relevance in predicting an individual’s risk for future atherosclerosis still remains an unexplored field.

Because of drastic individual variability in responses after cholesterol fat feeding no reasonable cholesterol tolerance test has been devised. Individuals also respond differently to single feeding and prolonged feeding. So, it still remains controversial whether single dose or a prolonged feeding programme could be a better screening procedure.

The possibility that the particular postprandial response of an individual to a single high cholesterol test load may be a determinant of the atherogenic process is the basis of the present work.

This work has been conducted on 26 male subjects suffering from diabetes, hypertension and ischemic heart disease.

The observed basal serum cholesterol was 232.45±52.35, 242.0±37.97 and 232.33±67.64 mg/dl in group A, B and C respectively.
All the groups had basal STC towards the upper limit for the age group as set by lipid research clinic for healthy males. The highest basal level was found in hypertensive subjects. This may be because of high consumption and their belonging to a better socio-economic status than diabetics and subjects suffering from ischemic heart disease (IHD).

The various Indian studies also support our finding of high basal STC in diabetics i.e. Mehrotra et al (1989) have found basal STC in 25 cases of diabetes (233.6±26.6 mg/dl). Mehrotra (1988) had found basal STC 278.50±50.47 mg/dl and Pant et al (1988) had found basal STC 259.88±44.79 mg/dl in diabetic subjects.

The Indian studies which support the high basal STC in IHD subjects were Das et al (1984), Wahal et al (1985), Mishra et al (1980), Shashikumar et al (1988), 249.0±70.14, 245.61±44.87, 249.8±58.8 and 257.40±29.33 mg/dl respectively.

On feeding a fall in STC was observed at the first hour in the diabetics and hypertensives but a rise was seen in IHD patients. At the third hour STC showed a rise over the first hour values in group A and B, but in group B the third hour value remained below the fasting value whereas in subjects of IHD there was a fall at third hour.

Thus on the basis of postprandial responses the studied subjects can be divided in three groups;
in one the subjects showed a fall at the first hour; in the second the subjects showed a rise at the first hour and the third who did not show any change.

In the past Nikkila et al (1982) and Richards, Havel (1957) also reported a fall in STC levels after prolonged feeding. The explanation for the fall could be related to the suppression of LDL receptors after overnight fasting (Medical clinics of North America Vol. 66 No. 2, March, 1982, p. 344). We hypothesize that the fat load is given; LDL receptors are stimulated by as yet some undefined hormonal or neurogenic mechanism in anticipation of the cholesterol load that will enter the circulation. Large amounts of LDL from intravascular compartment shifts within the intima and media immediately adjoining the intima resulting in an acute fall of serum LDL and STC level after one hour.

The increase of STC and LDL could not be explained with the help of existing literature, but our presumption is that the increase of STC and LDL might be due to decreased receptor activity either due to saturated receptors or a decrease in the number of receptors.

**HIGH DENSITY LIPOPROTEIN (HDL) CHOLESTEROL**

The basal levels were 40.58 ± 8.88, 45.1 ± 10.63 and 30.35 ± 8.98 mg/dl of group A, B and C respectively.
These levels were all within normal limits set by lipid research clinics. However, Group C had comparatively lower level of HDL than group A and B. The various Indian studies also support our findings of low serum HDL. Sasikumar et al (1988) and Wahal et al (1985) reported 37.6±6.42 and 36.67±10.13 mg/dl respectively whereas Shah et al (1980) reported lowest serum HDL in cases of IHD and acute myocardial infarction 21.8±7.2 and 21.7±6.4 mg/dl respectively in age groups of 40-50 years, while in studies on the diabetic subjects in the India the HDL level were higher than found in the IHD subjects. Bijlani et al (1984); Gahlot et al (1980) found 58.8±11.1 and 65.77±7.57 mg/dl of HDL in diabetic subjects respectively.

On feeding single high cholesterol test load no appreciable changes in HDL were observed at the first hour. At the third hour only group B (Hypertensives) showed significant rise in HDL level. Feeding induced rise in HDL in hypertensive was associated with fall in STC and LDL.

The outcome of this study, apart from high cholesterol test diet induced changes is that all the groups of patients showed higher ratio of STC/HDL (75) particularly in IHD patients. STC/HDL ratio
was more than 7 and lower percentage of HDL than other groups. The STC/HDL ratio and percentage of HDL are better predictors of atherosclerotic heart disease than the individual level of STC and HDL. The incidence of definite coronary event was inversely correlated to HDL. A cut off point for HDL has been reported by Bruner et al (1987) and is taken as 21% for male and 22% for female irrespective of age.

**SERUM TRIGLYCERIDE (STG)**

Basal STG levels were 210.80±68.90; 245.50 ±94.61 and 230.0±47.11 mg/dl respectively in group A, B and C. All the groups had higher STG value than set by lipid research clinics. Hypertensive had higher levels of basal STG than diabetics and IHD subjects.

After feeding single high cholesterol test load STG level showed a slight fall at the first hour in diabetics and hypertensive whereas a significant rise in IHD subjects and at the third hour diabetic subjects showed a significant rise, hypertensive subjects again showed a fall whereas IHD subjects showed fall from first hour but remained above the fasting values.

Barritt (1965) reported the peak of STG after seven hours in subjects of IHD after high fat diet. The fall in triglyceride could not be explained on the basis of present existing literature. For the fall in STG further evaluation is needed in future.

**VERY LOW DENSITY LIPOPROTEIN (VLDL) CHOLESTEROL**

Changes in VLDL were exactly similar to those observed in STG.

**LOW DENSITY LIPOPROTEIN (LDL) CHOLESTEROL**

Basal levels of LDL were 141.72±57.29, 139.40±36.0 and 157.58±63.64 mg/dl of group A, B and C respectively. These values were towards the higher level set by lipid research clinics. IHD subjects had higher LDL level than diabetics and hypertensives.

Feeding of single high cholesterol test load resulted in decrease in LDL levels at the first hour but the fall was insignificant. At the third hour LDL level started rising in diabetics, hypertensive and fall in IHD subjects. The explanation for the observed phenomenon is the same as that for STC.
LIPID RISK FOR ATHEROGENESIS

The lipid risk of the individual is variable; some individuals which were at high risk on basal lipid lipoprotein profile remained at high risk on postprandial level whereas some individuals which were at low risk basally become high risk on postprandial response; others showed a protected response.

However, this requires further work on a large scale for confirmation.