DISCUSSION
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CHANGE IN GROUP A

Changes in Serum Total Cholesterol (STC)

The fasting STC level in group A subjects was 194.51±53.07 mg/dl. There was a fall in this level 1 hour after ingestion of the test diet, and then a rising trend was noticed in the second and third hours.

On splitting the subjects into the two subgroups of IDDM and NIDDM, however, a marked difference was noticed in the fasting as well as all the postprandial values between the two, with the NIDDM patients having a markedly higher fasting STC concentration of 236.66±45.1 mg/dl, in contrast to the 158.38±25.22 mg/dl of IDDM patients, a difference that was statistically significant (p < 0.05).

One hour after ingestion of the test diet, this decreased to 219.66±29.16 mg/dl, a fall of 7.18 percent from the basal value; there was a progressive rising trend in the second and third hour values.

In marked contrast to this was the trend of STC in the IDDM subgroup, wherein the 1 hour postprandial value rose above the fasting level, and continued to rise at 2 and 3 hours, with the highest value being reached at 3 hours, to 175.95±14.69 mg/dl, a rise of 11.09 percent over the fasting level. The difference between the two subgroups at 1 hour was statistically highly significant (p < 0.02), and the differences at 2 and 3 hours were also statistically significant (p < 0.05).
Thus on the basis of post prandial response the diabetic population can be divided into three groups. Almost half the population (6 patients) showed a fall in STC concentration 1 hour after feeding, an equal proportion (6 patients) showed a rise in STC at 1 hour, and one patient showed no change from the fasting level at 1 hour.

In the past also, Nikkila et al (1962) and Havel (1957) have reported a fall in STC concentration after feeding. The explanation for this 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). When a high cholesterol load is given, LDL receptors are stimulated by some as yet undefined hormonal or neurogenic reflexes, in anticipation of the cholesterol load that will enter the circulation. Consequently, a large amount of LDL from the intravascular compartment shifts intracellularly, resulting in an acute fall in serum LDL and STC levels after 1 hour. The cholesterol levels slowly increase thereafter as a result of the absorbed cholesterol entering the circulation, and the reversal of movement of LDL that had entered the tissues earlier.

By the same reasoning, the rise in serum cholesterol in other patients could be explained by some inherent biochemical block in the above mentioned mechanism, in anticipating and assimilating the cholesterol load. The fact that IDDM patients were more prone to display this kind of behaviour suggests that aetiological factors of
IDDM play a dominant role in disruption of the above mentioned mechanism. But this is not exclusively the case, as shown by the fact that there were two patients in the IDDM subgroup, who showed a fall in STC, after 1 hour, and also there being two patients in the NIDDM subgroup who showed a rise in the 1 hour STC concentration.

Changes in HDL Cholesterol

The fasting and postprandial levels of HDL-c were within normal range in both IDDM and NIDDM patients and showed very little variation in the postprandial phase. Nor were there any marked differences between the two subgroups. Age, sex and smoking did not affect the HDL profile in any way.

Changes in LDL Cholesterol

A mean fasting LDL cholesterol concentration of 119.90±42.71 mg/dl was noted in the group as a whole, with a fall in the 1 hour value to 110.83±24.73 mg/dl followed by a slight rise at 2 and 3 hour intervals. The trend in LDL thus mirrors exactly the trend seen in STC concentrations. The same holds true for the trends of IDDM and NIDDM subgroups. Marked differences were observed in all the respective values, between the two subgroups, with NIDDM patients having considerably higher LDL-c concentrations. Except the third hour, all these differences were statistically significant (p < 0.05), with that at two hour being statistically highly significant (p < 0.02). Intracellular
movement of LDL in the immediate postprandial phase seems to be the explanation for this, as narrated earlier for STC. No age, sex or diabetic-control related differences was observed.

Changes in Serum Triglycerides (STG)

The fasting levels of STG in IDuM and NIDDM were 135.17±27.42 and 212.73±78.07 mg/dl respectively. Both IDuM and NIDDM patients showed a gradual and sustained rise in STG levels after ingestion of test diet, with the highest values being reached at 3 hours in both groups - the difference at 3 hours between the two groups being statistically significant. Brown et al (1961) and Angervall (1964) reported peak STG levels four hours after feeding in healthy subjects.

Treatment of diabetics with insulin results in decreased levels of triglyceride and VLDL, though the levels are still higher than in normal persons, as documented by Lewis et al (1972). The reason for this is the enhancement of lipoprotein lipase activity by the exogenously administered insulin. The fact that all the IDDM patients in our study were well controlled with insulin regimens could account for the finding of comparatively lower triglyceride concentrations in this subgroup.

Changes in VLDL

Changes in VLDL were exactly similar to those of STG.
CHANGES IN GROUP B

Changes in STC

The mean fasting STC was 159.32±48.34 mg/dl, the lowest amongst all the four disease groups. There was, however, a pronounced rise of 13.55% after 1 hour of diet ingestion, with marginally lower subsequent values - all postprandial values being above the fasting one. What was noteworthy was that all the eight patients in this group showed a rise in STC levels at the first hour. No studies as yet have been conducted on the postprandial lipid profile in these patients. Stigendahl et al (1984) have stated that there is reduced synthesis of endogenous cholesterol in cirrhotics. This could explain the low fasting STC levels in our patients, whereas the marked rise in STC levels after ingestion of exogenous cholesterol can be attributed to decreased clearance from plasma due to hepatic lipase inhibition, a finding reported by Schaefer et al (1985). Also decreased intake due to the marked anorexia in these patients could be another cause that requires quantification.

Changes in HDL Cholesterol

The HDL cholesterol levels increased after 1 hour but thereafter displayed only very marginal changes. The slight decrease shown by some patients at 2 hours appeared to be a transient phenomenon, as the values rose again at 3 hours; the two patients in whom the 3 hour level showed
a downward trend happened to be those whose 1 hour value of HDL-C had also stayed unchanged from the fasting level. We recommend further studies to explain the significance of the above finding in our study, as no explanation is forthcoming at present.

Changes in LDL Cholesterol

Five out of eight patients showed a rise in LDL-C at 1 hour, and all of these except one continued to show further rise at 3 hour.

In contrast, the remaining three patients who had a fall in LDL-C at 1 hour, in all these three, the downward trend continued till the third hour also, with the 3 hour values dropping below the basal level.

Thus two distinct trends could be appreciated, and we recommended further studies on larger groups of patients to elaborate further the cause of the same. Differences in hepatic LDL receptor activity could be one explanation.

Changes in Serum Triglycerides (STG)

Seven of the eight patients showed a gradual postprandial rise in STG, peaking at 3 hours. Schaefer et al (1985) have also reported increased levels of triglycerides, chylomicrons and VLDL in patients of primary biliary cirrhosis, and have attributed these abnormalities to hepatic lipase inhibition and altered cholesterol esterification.
Changes in VLDL

These were exactly similar to those in STG.

CHANGES IN GROUP C

Changes in STC

The highest fasting (295.02±76.83 mg/dl) as well as postprandial STC levels were observed in this group of patients, although the postprandial rise in STC was only slight (maximum 9% at 3 hours). Hyperlipidaemia is a well known feature of chronic renal insufficiency. Cholesterol ester turnover has been found to be raised together with triglyceride turnover in patients with the nephrotic syndrome (Mckenzie et al, 1968), and a possible relationship between cholesterol ester turnover and lipoprotein transport has emerged from the study of Nestel and associates (1968, 1970).

Our findings of only a very slight rise in STC in the postprandial phase finds support in the view of kayden et al (1963), that the composition of plasma cholesterol esters is only minimally affected by a single meal of a specific fat, but is readily influenced thereafter.

Changes in HDL Cholesterol

This was the only group in which the HDLC showed a marked rise from the basal (53.44±19.9 mg/dl) to the postprandial levels, rising to 74.42±38.75 mg/dl at 3 hour, a rise of almost 40 percent over the resting level. Since eight out of nine patients were of nephrotic syndrome,
understandably, the hyperlipidaemia associated with this condition has overtly influenced the overall mean HDL-c levels. Joven and Villawona et al (1990) stated that overproduction of lipoproteins containing apoprotein *s* is the principal cause of hyperlipidaemia in these patients, but Karadi and Romics et al (1989) found that serum lipoprotein (a) levels may be increased. The lone patient of CRF in this group had fasting and 3 hour HDL-c levels of 18 and 20 mg/dl respectively, much below the group mean. This conforms to the established finding of low HDL levels in CRF (Lewis et al, 1966, Rapoport and Aviram et al, 1978; Goldberg and Harter et al, 1983), although the reasons had not been defined. But recent studies show that low serum HDL concentrations in patients with CRF are related to decreases in the synthetic rate of apo AI/HDL (Martin rüh, C-Ming Lee et al, 1990).

**Changes in LDL Cholesterol**

The fasting level of 144.95±36.81, decreased gradually to 118.68±32.67 mg/dl after 3 hours. The marked progressive rise of HDL-c levels could be held responsible for affecting the derivation of LDL cholesterol values.

**Changes in Serum Triglycerides (STG)**

Hypertriglyceridaemia is a characteristic feature of nephrotic syndrome, as is apparent from the markedly high fasting and postprandial values, rising to 642.8±250.75 mg/dl at 3 hours, an increase of 33 percent over
the fasting value. This increase however, was not statistically significant due to the large standard deviation and small sample size. We recommend further studies with larger population groups to properly quantify the changes.

Murase et al (1975) provided evidence of a factor in uraemic plasma which inhibits lipoprotein lipase activity. This results in decreased clearance of triglycerides from plasma.

Changes in VLDL

Changes in VLDL are exactly similar to changes in STG.

CHANGES IN GROUP D

Changes in STC

Both the mean fasting and postprandial levels of STC were within normal range as established by Lipid Research Clinics with the postprandial levels showing only a minor and statistically insignificant rise. Studies in humans and primates have demonstrated that respiratory infections lead to lowered plasma cholesterol levels (Kerttula and Weber et al, 1988; Fiser and Denniston et al, 1972), since repeated respiratory infections are a feature of COPD, this could be one reason for the absence of any significant rise in STC levels in the postprandial phase.

Previously also, respiratory disease mortality has shown inverse association with cholesterol levels
(Kozarevic and McGee et al, 1981; Kagan and McGee et al, 1981; Neaton and Blackburn et al, in press). However, Smith and Shipley et al (1992), while confirming that such a relationship exists, argue that mortality studies do not allow for examination of the temporal relationship between the development of respiratory morbidity and plasma cholesterol level.

**Changes in HDL Cholesterol**

The mean fasting HDL cholesterol level was $38.33 \pm 11.09$ mg/dl which was almost unchanged for 2 hours after ingestion of the test diet, showing a slight decline at 3 hours to $35.97$ mg/dl. Thus, there was no significant change in the HDL cholesterol level.

**Changes in LDL Cholesterol**

The mean fasting LDL-c level was $129.59 \pm 37.61$ mg/dl. This showed a small rise of 9.27 percent at 1 hour, followed by very slight decreases at 2 and 3 hours. None of these changes were significant and no distinct trends could be identified.

**Changes in STG**

The most notable finding that emerged from our study of COPD patients was the sustained and sharp increase in STG concentration in the post prandial phase, while the fasting STG levels were within normal limits.
(90.39±24.74 mg/dl). The 1st, 2nd and 3rd hour mean levels showed a rise of 29, 42 and 55 percent respectively, over the fasting level.

This is the greatest magnitude of rise in STG in the postprandial phase seen in the four disease groups in this study. Even though the rise was not statistically significant. We believe that the relatively large standard deviation along with the small sample size could be responsible for the same, and therefore strongly urge that further studies with larger sample groups be carried out to elucidate the qualitative and quantitative significance of these changes.