SERUM LIPIDS IN ANXIETY NEUROSIS

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SUMMARY

Serum cholesterol, total triglycerides, HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol, free cholesterol and total phospholipids were studied in 36 patients of anxiety neurosis and 24 control subjects. Serum triglycerides, VLDL-cholesterol and free-cholesterol were found to be significantly raised while esterified cholesterol was significantly lowered in anxiety neurosis. A significant negative correlation was observed between the anxiety score and free cholesterol in female patients. The significance of these findings has been discussed.

Lipoprotein metabolism has gained profound importance ever since Miller and Miller (1975) reported the protective role of HDL cholesterol in the development of cororary heart disease (CHD). A number of recent investigations emphasize the relation of total cholesterol, total glycerides along with LDL cholesterol and VLDL cholesterol with CHD HDL cholesterol showing an inverse relation with the development of CHD is considered a major risk factor (Miller and Miller, 1975). More recently attention has been drawn to the apoprotein (carrier) rather than the lipid (carrier) component of serum lipoproteins, and the ratios of different lipoprotein are considered more sensitive indicators of atherogenesis than absolute values.

A number of studies provide considerable evidence for the role of anxiety and related emotional reactions in the development of CHD, seemingly more for angina pectoris than for MI (Jenkins, 1976). Although there is much work on lipid metabolism in cardiovascular disorders, It has been completely ignored in various stress conditions. The present study was therefore under-

taken to study the lipid pro-file of patients of anxiety neurosis.

METHOD

Thirty six patients of anxiety neurosis diagnosed as per Feighner's Diagnostic Criteria (Feighner et al., 1972) attending the out-patient Department of Psychiatry of the University Hospital (Banaras Hindu University, Vararasi) formed the sample of the present study. Patients who had been taking drugs during the past week or with concurrent physical illness were excluded. Each patient was subjected to a detailed evaluation: history of the present illness, past and family history of psychiatric illness and physical illnesses (diabetes, hypertension, CHD, renal diseases, thyrotoxicosis etc.), and personal history were recorded on a structured proforma. Apart from other details, personal history also included details of deitary habits, physical activity, smoking and alcohol intake. A physical examination and detailed mental status examination was done. Hamilton anxiety scale (Hamilton, 1969) was applied to measure the intensity of symptoms.

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Twenty four control subjects were selected from the students and employees of Institute of Medical Sciences, B. H. U. without evidence of any neuro-psychiatric or physical illness. Both patients and control were subjected to routine investigations to exclude any hepatic disorder, renal disease or diabetes mellitus.

About 6 ml of blood was collected after overnight fasting for 12 hours in the morning. Serum was stored at 6°C-4°C. All the analyses were done within two days of obtaining blood. Total cholesterol was determined by the method of Bowman and Wolf (1962) and free cholesterol was estimated by modification of Bowman and Wolf method (Bowman and Wolf (1962). The latter was subtracted from the former to get the esterified cholesterol fraction. Serum triglycerides were estimated by the method of Mendez et al. (1975). HDL cholestetol was determined after precipitation by phosphotungstate and magneisum chloride (Burstein et al., 1970). Then by applying the formula of Friedwald et al. (1972), LDL-cholesterol was determined.

RESULTS

1. Sample characteristics:

Anxiety neurosis patients were divided into different subgroups according to the Hamilton anxiety score. Nineteen patients were rated mild (score< 13) whereas rest seventeen belonged to the moderate group (score between 13 & 24). However there was no case of severe anxiety (score>25). Eight patients of mild anxiety, seven of moderate group and six of the control subjects belonged to female sex. The age group distributions of the patients as well as controls is given in table I.

2. Lipid profile of control group (Table II):

Total cholesterol concentration was found to be in the range of 145-254

TABLE I. Distribution of cases and control subject in different age groups

| Age in years | Contro | ol group | Anxiety neurosis | | |
|---------------|--------|----------|------------------|----------|--|
| | N | % | N | <u>%</u> | |
| 10—29 | 10 | 41.7 | 19 | 52.8 | |
| 30—4 9 | 10 | 41.7 | 17 | 47.2 | |
| 50 or above | 4 | 16.6 | _ | | |

mg/dl. The mean triglyceride concentration was 90.16+25.45.mg/dl. These values were comparable to the published reports from India (Bhattachariee et al., 1978; Barrington et al., 1980; Gandhi, 1982). Mean serum HDL cholesterol concentration was 47.80 ± 7.74 mg/dl ranging from 40.9 to 58.8 mg/dl except one male having 38.3 mg/dl and one female having 69.0 mg/dl. In general females were found to have higher HDL cholesterol level in comparison to males. The LDL-cholesterol (LDLC) ranged between 67.4-177.4 mg/dl. The mean serum VLDL-cholesterol was 17. 99+5.08mg/dl and the range was from 12.7 to 26.6 mg/dl.

Mean serum free cholesterol level was 54.25 ± 12.96 mg/dl and that of esterified cholesterol was 135.53 ± 26.65 mg/dl, quite within the normal range of 70-80% of esterification of cholesterol.

3. Lipid changes in Anxiety Neurosis (Table-II):

The mean serum total cholesterol concentration (of both subgroups) in anxiety neurosis did not show any significant difference whereas their total glycerides showed highly significant increase with respect to controls. Similarly mean serum HDL cholesterol and LDL cholesterol concentration of anxiety neurosis/neurotics did not show any change whereas there was marked increase in their VLDL concentration.

| | | T _e | Tg mg/dl | HDL _e | LDL _c | VLDL _c | F _c mg/dl | E _e |
|-------------------------------------|--------------|----------------|-------------|------------------|------------------|-------------------|-------------------------|----------------|
| | | | | | | | | |
| Control | Mean | 189.78 | 90.16 | 47.80 | 123.98 | 17.99 | 54.25 | 135.53 |
| (N=24) | SD. | 30.63 | 25.45 | 7.74 | 0.87 | 5.08 | 12.96 | 26.65 |
| Mild anxiety neurosis @ (112) | Mean | 181 .80 | 127.42*** | 47.08 | 108.92 | 25 .92 *** | 64.88 | 112.50** |
| (N=19) | \$D . | 32.02 | 36.95 | 9.98 | 31.18 | 7.39 | 18.36 | 27.45 |
| Moderate anxiety neurosis @ (13-24) | Mean | 178.83 | 123.96 | 45.81 | 108.22 | 24.78*** | 56.13 | 119.75* |
| (N ⇒17) | SD. | 31.18 | 34.04 | 6.26 | 30.66 | 6.87 | 15.21 | 22.84 |

TABLE-II. Lipid profile in control and anxiety neurosis patients.

Free cholesterol was high only in mild anxiety neurosis patients while esterified fraction showed significant decrease, both in mild and moderate anxiety neurosis. The ratios of HDL cholesterol to total cholesterol and HDL cholesterol to LDL cholesterol did not show any significant change in comparison to controls.

4. Correlation of anxiety scores (indicating degree of stress) with the different lipid parameters was studied by drawing regression line. No significant correlation was found with any of the parameters. However, when male and female groups were separated, though no correlation was obtained in male patients, significant decrease in free cholesterol with increasing degree of anxiety score as measured by the hamilton Anxiety Scale was seen in female patients of anxiety neurosis (r=0.678).

DISCUSSION

In the present study the mean level of total cholesterol did not show any change but there was significant rise of total glycerides in both mild and moderate anxiety neurotics. The rise in glycerides may be due to increased activation of autonomic nervous system both in acute as well as in mild and prolonged, emotionally induced stress situations. During stress there is release of adrenocorticotrophic hormone (ACTH) and corticosteroids along with discharge of catecholamines which are known for their lipolytic action. Circulating corticosteroids suppress the availability of glucose in adipose tissue by antagonising the action of insulin. Thereby the lipolytic activity predominates in adipose tissue leading to increase in circulating free fatty acids, which get esterified to triglycerides in tissues like liver due to the presence of the enzyme glycerokinase. Similar increase in triglycerides has been observed by other workers in différent stress conditions (Wolf et al., 1962; Taggart and Carruthers, 1971). Wolf et al. (1962) found high risk in triglycerides and total cholesterol in people exposed to difficulties in family or job including stressful interviews. whereas Taggart and Carruthers (1971) observed increase in triglycerides level reaching peak after one hour of the event

^{*}P<0.05; **p<0.01; ***p<0.001 @Hamilton Anxiety Score.

in racing drivers. In the latter case stress was more of acute and transitory type whereas our patients had a prolonged continuous stress of varying degrees.

High level of serum triglycerides has been reported in coronary heart disease (CHD) (Dutt, 1967; Bhattacharjee et al., 1978; Kaukola et al., 1980). It has been shown that hypertriglyceridemia is more frequently associated with atherosclerotic cardiovascular disease than hypercholesterolemia (Carlson, 1960; Hayes and Neil, 1964; Carlson and Bottiger, 1972; Rhoads et al., 1976) and also acts as an independent significant major risk factor for coronary heart disease.

Significant increase in VLDL choesterol concentration was in patients of anxiety neurosis. It is well reported that plasma VLDL is atherogenic (Goldstein and Brown, 1977) and hyper VLDL-cholesterolemia has been reported by différent workers in ischaemic heart disease (Vergani et al., 1978; Barrington et al., 1980)

In the present study a significant risk in free cholesterol in mildly anxious patients and more significant reduction in esterified cholesterol was noted, whereas moderate anxiety neurotics had only a significant reduction in esterified cholesterol fraction. The underlying mechanism is difficult to explain. To know the cause of decreased esterification the activity of serum lecithin cholesterol acyl transferase (LCAT), the major cholesterol esterifying enzyme, should be determined.

No significant correlation was observed between the different lipid parameters studied and anxiety score from Hamilton anxity scale. This may be due to the fact that no single scale is ideal for measuring stress of a person and all scales available, to date, tap trait anxiety in addition to symptoms of an-

xiety. Among female patients however, a definite negative correlation of free cholesterol with increase of anxiety score was obtained.

In conclusion, patients of anxiety neurosis showed hypertriglyceridemia and increased VLDL-cholesterol concentration with reduction in esterified cholesterol level. The first two changes favour the development of atherosclerotic process.

The findings of the present work suggest that anxiety neurotics in the long run are at a greater risk for the development of atherosclerosis and its cardiovascular complications, and that triglycerides, VLDL cholesterol and esterified cholesterol are perhaps the biochemical variables mediating the cardiovascular psychosomatic response. These assumptions are however tentative and further work is needed.

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