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Research Article

A Comparitive Study of Serum Lipid Profile of Suicide Attempters Versus Normal Age, Sex and BMI Matched Controls

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ABSTRACT

The relation between the biology of lipid and the psychobiology of suicide attempt is demonstrably an important scientific and public health tissue. The present study was conducted to evaluate the use of serum lipid profile as a peripheral marker of suicidal risk.

The serum lipid profile of 30 suicide attempters were compared with 30 age, sex and BMI matched normal healthy controls. Instruments used were Risk Rescue Rating Scale, Hamilton Depression Rating Scale, General Health Questionnaire and calorimetric method of lipid estimation. Diagnosis was based on DSM-IV.

Comparison of serum total cholesterol, HDL, LDL, TGL and VLDL between the study and control groups showed no difference. Correlation analysis of RRRS, HDRS and serum lipid profile did not show any significant relationship. Implications of the above findings are discussed.

Key words: *suicide, depression, cholesterol, lipid profile*

INTRODUCTION

Primary prevention trials, which have shown that lowering of serum cholesterol concentrations in middle-aged subjects by diet, drugs, or both leads to a decrease in CHD have also reported an increase in deaths due to suicide or violence. In a review of 6 randomized controlled, primary prevention trials, Muldoon et al (1990) found that lowering of raised serum cholesterol in middle-aged subjects by diet, drugs, or both was associated with significant decrease in deaths from CHD but not in overall mortality. There was significant increase in mortality due to suicides or violence; compared with control group, the treated groups had 28 fewer deaths from CHD and 29 more deaths from suicide, homicide and accident.

Vikkunen (1979, 1983 & 1984) found that subjects with antisocial personality disorder and aggressive conduct also have lower blood cholesterol than did control groups. There was no difference in concentration of triglyceride level. Monkeys on a diet low in saturated fat and cholesterol diet were significantly more aggressive than were control animals on a normal diet (Kaplan, 1990).

Patients in psychiatric hospitals who had higher cholesterol concentrations (mean 7.55mmol/l) were less regressed and withdrawn than were those with lower concentrations (mean 4.8mmol/l) although nutritional status was same (Sletten, 1964). Higher serum cholesterol levels were also found in firemen and male food market workers who had stable personality traits than in matched controls (Jenkins, 1969). In contrast, no relationship between serum cholesterol and mortality either from accidents or suicide has been discovered in community cohorts (Zureik et al, 1996).

In two large randomized double blind trials the lipid research clinic coronary primary trial (1984) and Helsinki Heart study (Frich et al, 1987) the benefits of a decrease in CHD mortality were offset by increase in deaths from accident and violence.

Maes et al (1994) found HDL/cholesterol ratio to be significantly lower in subjects with major depression and in patients with depressed mood who had at some time made a medically serious suicide attempt.

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Collectively including over 2000 patients, 5 of the 6 studies showed an association between low cholesterol and suicidal ideation or attempt (Muldoon et al, 1990 & 1992; Lipid Research Clinics Programme 1984; Frich et al 1987). Vikkunen (1985) had suggested that increased mortality due to accident and violence in groups with low cholesterol might be associated with low serum serotonin turnover in brain.

But there have been studies which do not support the association between low cholesterol and suicidal behaviour (Smith et al, 1990; Law et al, 1994; Zureik, 1996). Randomized control trials using cholesterol-reducing treatments have found no evidence of excessive risk of suicide (Scandinavian Simvastatin Survival study group, 1994).

Thus the relationship between serum cholesterol and suicides has been quite obscure and controversial. But the implications of this causal relationship have far reaching consequences in screening for high-risk suicidal individuals and in decreasing total mortality.

MATERIAL AND METHODS

Sample

The sample is comprised of two groups-

- Suicide attempters → Attempters admitted to Medical College hospital, Kozhikode satisfying the inclusion and exclusion criteria.
- Control group → Normal healthy volunteers matched one to one for sex, group matched age (group consists of 5 years), and BMI.

Inclusion criteria

- Age between 17 to 55 years
- Informed consent
- Belonging to either gender
- Fulfilling criteria for suicide attempt (for study group)
- Able to converse in Malayalam / English

Exclusion criteria

- Presence of major medical illnesses that can produce hyperlipidemia
- Use of drugs that can produce hyperlipidemia
- Burns
- Pregnancy
- Substance abuse including cigarette smoking
- GHQ score above 6 (for controls)
- Mental retardation
- Delirium
- Anorexia nervosa

PROCEDURE

Informed consent was taken from all subjects participating in the study after they satisfied the inclusion and exclusion criteria. They were then divided into two groups – a study group consisting of 30 suicide attempters and a control group consisting of 30 - age, sex and BMI matched normal healthy controls. Relevant investigations were done when indicated. All assessments were done within a week of the attempt. Psychiatric diagnosis was based on DSM IV criteria (APA, 1994). Severity of suicide attempt was assessed by Risk Rescue Rating Scale (RRRS) (Weisman and Worden, 1972). The severity of depression was rated using Hamilton Depression Rating Scale (HDRS) (Hamilton, 1967). General Health questionnaire (GHQ-Goldberg, 1972) was administered to the Control group to screen out psychological problems.

All subjects were kept fasting over night for 12 hrs and 5 ml of blood was drawn from the cubital vein next morning using disposable syringes. Blood was collected in non-oxalate test tube and sent within 2 hours for biochemical analysis. Sample was immediately centrifuged and serum total cholesterol, HDL, LDL, TGL and VLDL was estimated by enzymatic calorimetry (Ciba Corning's Express plus Autoanalyser).

ANALYSIS

Total cholesterol, triglyceride, HDL, LDL, VLDL were compared among the study group, control group, depressed and non-depressed suicide attempters. Association of lipid profile with socio-demographic variables, RRRS and HDRS was evaluated. Parametric and non-parametric variables were compared using 't' test and Chi-Square test (with Yate's correction). Correlation between variables was done using Pearson's Correlation Coefficient.

RESULTS

The mean age of the study group (27.03+8.7, range 17-46yrs.) and the control group (27.6+8.5, range 17-55yrs.) were comparable. There were 11 males and 19 females in both groups each. The mean BMI of the study group (20.2+3.9) and the control group (20.16+3.5) were also comparable. There was no statistically significant difference in mean total cholesterol, mean HDL, mean TGL, mean VLDL between the two groups (Table1).

The commonest psychiatric diagnosis in the study group was adjustment disorder (53.33%) followed by major depression (33.33%), schizophrenia (3.33%), delusional disorder (3.33%) and personality disorder (3.33%). 6.67% of the study group had no diagnosable psychiatric disorders.

Comparison of the lipid profile between the depressed suicide attempters and non-depressed suicide attempters showed no significant differences (Table 2).

Comparison of lipid profile of depressed suicide attempters and age and sex matched controls showed no significant differences (Table 3). Comparison of lipid profile between non-depressed suicide attempters and the age and sex matched control group also did not show any significant difference (Table 4)

The correlation between the RRRS and total cholesterol (r= -0.11, NS), RRRS and HDL (r= -0.01, NS), RRRS and LDL (r= -0.13), RRRS and TGL (r=-0.02, NS), and RRRS and VLDL (r= -0.02, NS) did not show any significant relationship. Similarly the correlation analysis between HDRS and total cholesterol (r= -0.13, NS), HDRS and HDL (r= -0.13, NS), HDRS and LDL (-0.19, NS), HDRS and TGL (r= -0.19) and HDRS and VLDL (-0.29, NS) did not show any significant relationship. The correlation between RRRS and HDRS also did not show any significant relationship (r=0.33, NS).

DISCUSSION

The debate about possible adverse effects associated with low or lowered serum cholesterol has raised important scientific questions concerning links between lipids and behaviour.

Increased mortality from suicides, accidents and violence in studies designed to reduce prevalence of acute myocardial infarction by lowering serum cholesterol was an unexpected finding. Primary prevention studies show the number of violent deaths to be equivalent to number of lives saved from acute myocardial infarction, thereby eliminating any improvement in overall mortality. It has however required metanalysis of several studies involving over 20,000 patients to demonstrate this effect. These studies have been reviewed by Muldoon et al (1993).

Generally patients entered in studies have cholesterol levels about 6.5mmol/l and a 10% reduction would lead to a level of 5.8mmol/l, which doesn't represent hypocholesterolemia. The question raised with this finding is whether low or lowering serum cholesterol causes violent deaths. Our study showed no relationship between hypocholesterolemia and attempted suicide. However, since we did not have baseline cholesterol of these individuals we did not know the change in cholesterol levels with the attempt. If there was a dip then 'lowering serum cholesterol' may therefore be more important than intrinsically low levels. Therefore a longitudinal study with serial lipid profiles assessed before and after the attempt could better substantiate these observations.

A study done by Partonen et al (1999) on a total of 29,133 men aged 50-69 years followed up for 5-8 years concluded that low serum cholesterol was associated with low mood and subsequently a heightened risk of major depressive disorder and death from suicide. Thus he concluded an indirect link between serum lipids and suicide via depression. Two studies (Oxenkrug et al. 1983; Yates & Wallace 1987) have found normal levels of cholesterol in depressed patients and normal population. But two other studies (Maes et al. 1994; Glueck et al. 1976) have reported lower cholesterol than normals. Also low plasma cholesterol could well be the effect rather than the cause of depression. Decreased appetite and weight loss from depression could lead to low cholesterol. Another possible association is for a threshold effect of cholesterol on depression rather than a linear association. This is because longitudinal studies have shown consistent findings only at very low cholesterol levels (4-5mmol/l). In our study comparison of depressed and non depressed attempters show lower mean cholesterol for the former but it failed to reach a

statistical significance. The same findings were seen when the depressed attempters were compared with the matched control group. However, one must note that the number of depressed patients were only 9, i.e., 30% of the total attempters. However weak or strong the association the small sample size alone limits any generalisation of these findings.

There are studies linking personality traits with lipid levels. Vikkunen (1979) and Freedman et al (1995) showed that antisocial personality disorder had lower mean cholesterol levels compared with other types of personality. New et al (1994) studied 94 subjects and found that borderline personality disorder patients had low levels of cholesterol compared with normal controls and they were correlated with increased impulsivity. A personality profile was not assessed in our study and this could have thrown more light into the relation between serum lipids and impulsive suicidal attempts.

Another area this study addressed is to find the correlation between the risk rescue score, HDRS and depression. The study by Verma et al (1999) had found a negative correlation between risk rescue score and serum lipid profile. However, in our study we found no such correlation.

One factor to be kept in mind is that the suicidal act or the cause (stressor) is associated with the considerable amount of autonomic arousal. This leads to increased catecholamine levels, which in turn could alter serum lipid profile. Some studies report such alterations for nearly 2 months after the major stressor. There is no way of calculating by how much and for how long the lipid levels are altered. This could be a significant confounding factor as altered lipids levels could well be the result rather than the cause of the suicidal attempt.

One difficulty with this study is that in the brain, endogenous synthesis appears to be the main source of cholesterol and uptake of LDL by the brain from the plasma is low. The question is whether peripherally altered lipid levels do actually cause altered levels inside the brain. If so whether all the five parameters in the lipid profile or only some of them. If the peripheral serum lipid profile cannot be used as a marker for suicidal risk then probably the brain lipid profile would have to be used and an adequate peripheral representation of this would be needed.

The relation between the biology of lipids and psychobiology of suicidal attempt is demonstrably an important scientific and public health issue. It also has implications in the implementation of new treatment and preventive strategies that should include a careful evaluation of not only the physical consequences but also the psychological consequences of lowering serum lipid levels.

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