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Effects of Mental Stress on Serum Triglyceride Level

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Abstract: To investigate the effect of mental stress on serum triglyceride concentration. This was a case control study. Subjects were 100 patients with hypertriglyceridemia and 100 healthy without hyperlipidemia. A self-administered questionnaire assayed the exposure to stress and anxiety in the past 6-12 months with the six point likert scale. Data were analyzed by SPSS software using chi-square, independent sample t-test and Pearson's tests. For all analyses, alpha level was 0.05. Mean of stressful life events in case group was 16.87±10.39 and in control group was 4.15±2.25. There was a significant difference between two groups ($p<0.001$). The results indicate that the stressors in the past 6-12 months may elevate the level of triglyceride. Therefore, hypertriglyceridemia is one possible mechanism of ischemic heart disease in stressed people.

Key words: Triglyceride, stress, case-control study, anxiety, concentration, hyperlipidemia

INTRODUCTION

Most people throughout the world die from the consequences of cardiovascular disease (Csef and Hefner, 2005). The total cholesterol and LDL-C levels are correlated with a high Coronary Heart Disease (CHD) risk even in people over 80 (Li *et al*., 2004). Specific therapeutic approaches aimed at reducing and coping with stress may, in future, help prevent diseases of the heart and lower the risk of contracting a myocardial infarction (Csef and Hefner, 2005). Substantial evidence indicate that risk for developing coronary heart disease rise with elevated level of total cholesterol, triglycerides and LDL cholesterol and with lower levels HDL cholesterol. Additionally, there has been much interest in possible influences of behavioral factors on serum lipids and lipoproteins (Bachen *et al*., 2002). There is a large body of research addressing the consequences of stress coping on blood lipid concentration (Gerhard *et al*., 2003). Numerous studies have shown the relationship between lipid concentration and mental stress (Bacon *et al*., 2001; Patterson *et al*., 1993; Podbevsek, 2005; Bijlani *et al*., 1986; Muldoon *et al*., 1995; Patterson *et al*., 1995). Research of the last decades has shown lipid changes during stressful experiences. Severe forms of real or perceived stress do appear to alter lipid levels. Acute laboratory stress is frequently associated with short-term alterations in lipids and lipoproteins (Niaura *et al*., 1992).

Acute mental stress can produce rapid elevations in serum cholesterol concentration. It can also increase hemoglobin concentration and hematocrit. Therefore, increases in serum cholesterol level after acute mental stress are analogous to those with standing and may reflect hemoconcentration rather than altered lipoprotein metabolism (Muldoon *et al*., 1992).

Patterson and coworkers found Significant increases for total cholesterol, triglycerides, high density lipoprotein cholesterol, low density lipoprotein cholesterol and free fatty acid (all ps<.05) during stress (Patterson *et al*., 1995).

Type a component behavior and other aspects of personality, appear to be associated with an atherogenic lipid profile (Niaura *et al*., 1992). Vogele found that high hostiles had higher fasting triglyceride and VLDL-c levels than low hostile subjects (Vogele, 1998).

Lipid and lipoprotein changes have been attributed to the effect of epinephrine on lipoprotein lipase, hepatic lipase and hormone-sensitive lipase activities. The overall effect of epinephrine action on these enzymes is to increase fatty acid efflux from adipose tissue (Fur *et al*., 1999). Lipolysis is also stimulated by mental stress through ß-adrenoceptors (Stallknecht *et al*., 2001).

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MATERIALS AND METHODS

Participants were 100 patients with hypertriglyceridemia that the level of triglyceride was more than 400 mg% without family history of hypertriglyceridemia and the control group was 100 healthy subjects without hyperlipidemia.

All subject fasted for 14 h and blood lipids were measured in serum before breakfast. On arrival the subjects were seated in a reclining arm chair for 10 min before taking samples. Care was taken not to apply venous obstruction longer than 3 min. Serum samples were separated after 10 min centrifugation and stored at refrigerator at 2-8°C until assayed and analyzed on 48 h. A self-administered questionnaire assayed the exposure to stress and anxiety in the past 6-12 months with the 6 point likert scale. It consists of 45 important life events that lead to stress.

Data were analyzed by $SPSS_{12}$ software by chi-square, independent sample t-test and Pearson's tests. For all analyses, alpha level was 0.05.

RESULTS

The subjects were 114 male (57%) and 86 female (43%). In case group there was 9 singles and 91 marriages and in control group there was 11 singles and 89 marriages. The mean of age in case group was 47.7±9.6 and in control group was 48.2±9.16.

The mean of anxiety in male was 29.48±9.7 and in female was 29.825±10.09 (p>0.05). The mean of stressful life event in male was 11.13±10.56 and in female was 9.6±8.7 (p>0.05).

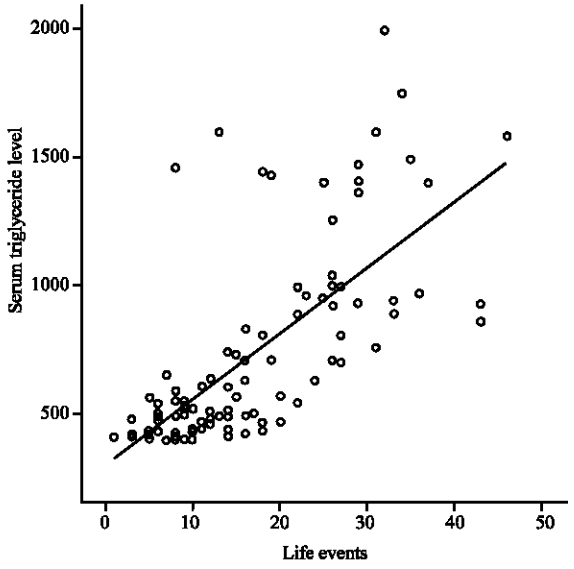

Fig. 1: The relationship between triglyceride level and life events

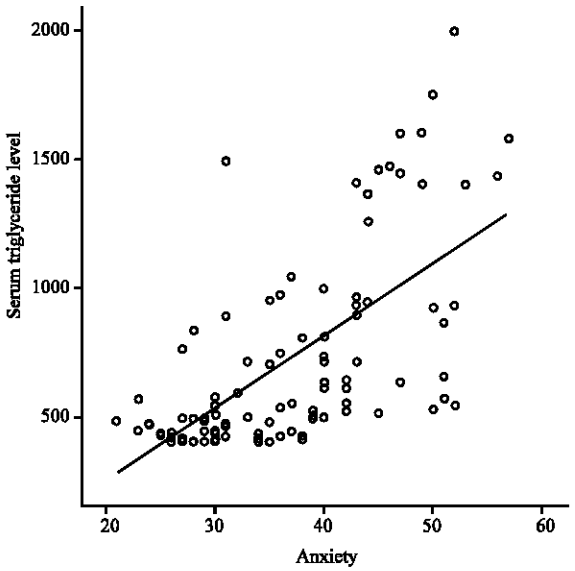

Fig. 2: The relationship between triglyceride level and anxiety

Mean of stressful life events in case group was 16.87±10.39 and in control group was 4.15±2.25. There was a significant difference between two groups (p<0.001).

The mean of anxiety in patients with hypertriglyceridaemia was 37.2±8.66 and in control group was 22.06±2.4. There was a significant difference between case and control group (p<0.001).

There was a significant relationship between triglyceride level and life events (r = 0.7 and p<0.001) and anxiety (r = 0.642 and p<0.001) (Fig. 1and 2).

DISCUSSION

The goal of the present study was to assess the effect of stress on concentrations of triglyceride. It is now established that laboratory-administered psychological stressors of only 5 to 30 min duration, such as common mental arithmetic and speech tasks, reliably elicit small increases in serum cholesterol and triglycerides and larger elevations in free fatty acids (Bachen *et al*., 2002).

Consistent with many earlier reports, the present study found a significant relationship between mental stress and serum lipid level (Bachen *et al*., 2002; Gerhard *et al*., 2003; Bacon *et al*., 2004; Patterson *et al*., 1993, 1995; Podbevsek, 2005; Bijlani *et al*., 1986; Muldoon *et al*., 1995). Most of studies explain the mechanism of this relationship.

A review by Brindley and Rolland discussed some metabolic and endocrine changes that can be associated with a stress type of metabolism, diabetes, obesity, hypertension, smoking and the consumption of diets rich in fat and refined sugar, or poor in acerbate. These are some of the risk factors associated with premature atherosclerosis, coronary thrombosis and stroke. It has been proposed that an increased control of metabolism by the 'stress' or counter-regulatory hormones, relative to insulin, is a common feature of these risk factors. Particular emphasis was placed upon the action of the glucocorticoids which can produce insulin insensitivity, leading to hyperglycemia, hypertriglyceridaemia, hypercholesterolemia and hyperinsulinaemia. Furthermore, glucocorticoids can decrease energy expenditure and, together with insulin, promote energy deposition. These observations provide a partial explanation for the metabolic changes that can accompany the risk factors and clarify why they interact in promoting atherosclerosis (Brindley and Rolland, 1989).

Circulating free fatty acids are an energy substrate and levels increase with sympathetic activation, as occurs during stress. However, free fatty acids may be part of a chain of metabolic events that leads to hypertriglyceridaemia and elevated VLDL, may have deleterious effects during myocardial ischemia and may mediate insulin resistance and the development of type 2 diabetes (Bachen *et al*., 2002). In lean subjects, mental stress stimulates glucose uptake and energy expenditure and produces vasodilatation; activation of ß-adrenoceptors is involved in these responses; and in obese patients, the effects of mental stress on glucose uptake and systemic vascular resistance, but not on energy expenditure, is blunted (Seematter *et al*., 2002).

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