THE STATUS OF SERUM LIPID PROFILE IN STRESS-RELATED PRE-INSOMNIA

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Article Received on 03/12/2014                Article Revised on 24/12/2014             Article Accepted on 15/01/2015

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ABSTRACT
Understanding of the correlation between stress, pre-insomnia and serum lipid profile status may provide clues toward the management of stress-related cardiovascular diseases. This study assessed the serum lipid profile on a total of thirty seven (37) male and female subjects; twenty eight (28) of which are students selected from different departments and nine (9), within Kano city. Twenty five (25) served as experimental subjects and twelve (12) as control subjects. Their blood pressure was taken and information on general life style, sleeping as well as reading habits was recorded by means of well-structured questionnaire prior to sample collection. Venous blood (5ml) was collected from each subject, centrifuged and the sera stored. Serum lipid profile was assayed using commercial kit from fortress diagnostics limited. When the Mean serum Total Cholesterol, High Density Lipoprotein Cholesterol, Low Density Lipoprotein Cholesterol and Triglyceride levels of test subjects were compared with those of the control subjects the difference was not significant (p>0.05). When comparison was made within the study group, based on borderline risk of high and normal blood pressure, the serum lipid profile level was found to differ significantly (p<0.05). Thus our study showed that acute sleep deprivation (pre-insomnia) does not alter serum lipid profile, however, is associated with mild hypertension which is a risk factor for cardiovascular diseases.

KEYWORDS: Pre-insomnia, Stress, Lipid profile.
1.1 INTRODUCTION

Insomnia is a subjective feeling of having difficulty initiating, maintaining or restoring sleep, or having poor sleep quality (Roth, 2007). It was reported to be associated with substantial impairments of an individual’s quality of life (Leger et al., 2012), depression (Sarsour, et al., 2010) and accident occurrence (Chiang, et al., 2012). Insomnia is the most common sleep disorder, affecting one in four people (Kessler 2011; Shatzmiller 2012; American Academy of sleep medicine 2001). Many people may not realize that insomnia and short sleep duration correlate with various health problems including cardiovascular disease, anxiety, and potentially cancer (Terauchi 2012; Chayon 1998; Kakizaki 2008; Verkasalo 2005; Philips 2007). Insomnia also increases mortality in adults (Chien, 2010 and Hublin 2011). Although the cause and physiology of insomnia have not been completely understood, it is generally considered as one kind of hyperarousal disorder related to increased activity of hypothalamic pituitary adrenal-axis (Roth, 2007). During this process cortisol (Vgontzas et al., 2001) will be secreted excessively. Elevated cortisol is able to induce higher cholesterol (Fraser et al., 1999).

Pre-insomnia (onset of insomnia) may be a consequence of another underlying medical problem e.g. mental health issue, physical health issue, hormonal imbalance, medication, and stimulant. Recently, it was demonstrated that paradoxical sleep deprivation for four days has significant effect on lipid metabolism such as increasing Low Density Lipoproteins (Anderson et al., 2004). In addition, cholesterol concentration was significantly decreased in rats exposed to cold environmental temperatures, indicating increase corticosterone synthesis and activation of HPA system (Cvijic, Davidovic and Jordjevic, 2003). Recent study suggested that short sleep duration was associated with higher risks of hypercholesterolaemia in the adults of U.S (Gangswisch et al., 2010) and japan (Kaneita et al, 2008) likewise reduce sleep duration was found to be related to dyslipidemia in other studies (Chen, and Zhang, 2014).

During the past two decades, considerable evidence has accumulated showing an association between cardiovascular disease (especially adverse coronary artery disease) and markers of stress and other psychological factors, such as anxiety, depression and somatization (Artham et al., 2008; Hemingway and Marmot, 1999; Rozansk et al., 2005). Recent studies have shown that disturbed sleep and sleep deprivation are associated with subsequent occurrence of cardiovascular alterations (Stickgold and Walker, 2007; Cintra et al., 2005; Perry et
al., 2007), behavioural changes (Anderson et al., 2005; Spiegel, Sheridan, and Van Cauter, 2002; Andersen et al., 2006) as a result of weight loss; however, unlike the decrease in LDL, the reduction of triglyceride concentration is not related to this weight reduction (Perry et al., 2007). Another ones have shown that habitual sleep duration <5hr per night is associated with an increased prevalence of arterial hypertension (Gangwisch et al., 2006; Gottlieb et al., 2006; Cappuccio et al., 2007). Some articles have proposed that activation of the sympathetic nervous system by sleep deprivation may be involved in triggering cardiovascular events in the morning hours (Tochikubo et al., 1996 and Lusardi et al., 1999).

A review reported by Dimsdale and Herd showed that the level of free fatty acids and total cholesterol increases in acute and chronic stress (Dimsdale, 1982), which may be attributed to insomnic condition. Similarly a study conducted by Fakhari and colleagues in 2004 demonstrated increased triglyceride in individuals who had experienced high levels of stress in the preceding 6-12 months (Fakhari et al., 2007). This may be attributed to the finding that cortisol causes the mobilization of fats (lipids) deposited in the adipose and other tissues, leading to an increased levels of lipids in the blood (Bower and Sergerstrom, 2001). More recently, our previous work on stress has shown that the higher the examination stress the higher the serum Cholesterol, Low Density Lipoprotein Cholesterol and Triglycerides levels and lower the serum High Density Lipoprotein levels, suggesting an increased risk of hyperlipidaemia in stress condition (Uba et al., 2014). The response to examination stress may be related to the enhanced utilisation of cholesterol in the andrenal cortex for steriodogenesis (Bijlani et al., 1986). Increase catecholamine levels result in up regulation of lipoprotein lipase, leading to increased concentrations of free fatty acids (FFA) in the serum that are transformed by the liver into cholesterol and triglycerides. Cortisol, on the other hand, increases the deposition of abdominal fat, which is more sensitive to lipolitic agents and related to insulin resistance (Resmond, 2005), elevated lipid profiles in turn may mediate longer term deleterious effects on physical health including heart disease and stroke.

The studies cited above have not been able establish proper correlation between stress, pre-insomnia, lipid profile and cardiovascular disease. This study is aimed at determining serum lipid profile status in pre-insomnic individuals in stress condition. The specific objectives are (1) To find if pre-insomnic condition is enough to cause lipid profile alteration (2) To observe whether an acute insomnia (pre-insomnia) is the risk factor for or the consequence of lipid profile alteration.
2.1 MATERIAL AND METHOD

2.1.1 Study Site, Subjects and Sample Collection
The study was carried out at Bayero University, Kano, Department of Biochemistry. A total of thirty seven (37) male and female subjects; twenty eight (28) of which are students selected from different departments and nine (9), within Kano city. Twenty five (25) served as experimental subjects and 12 as control subjects. Their blood pressure was taken and information on their general life style, including dietary habit was recorded by means of structured questionnaire prior to sample collection. Venous blood (5ml) was collected from each subject, centrifuged and the sera stored.

The study was approved by the ethical committee of the institution and the informed consent was obtained from the study subjects.

2.1.2 Methodology

2.1.2.1 Estimation of Serum Lipid Profile
Serum total cholesterol was assayed by Tindar’s reaction (Evans and Stein, 1986; NIH, 1990), Serum Triglyceride level was determined as described by Fossati and Precipe (1982), HDL-C, using commercial kit from fortress diagnostics limited and the serum LDL-C level was calculated by Friedwald’s formular (Friedwald et al., 1972).

2.1.3 Statistical analysis
The result were expressed as means ± standard deviation. One – way analysis of variance ANOVA was used to analyse Lipid Profile data followed by Student T-Test to calculate statistical significance difference between the various groups.

The study was approved by the ethical committee of the institution and the informed consent of the study subjects was obtained prior to experimentation.
3.0 RESULT AND DISCUSSION

3.1 RESULT

Table 3.1: Mean ± standard deviation of serum total cholesterol, HDL-cholesterol, LDL-Cholesterol, and Triglyceride

<table>
<thead>
<tr>
<th></th>
<th>TC (mmol/l)</th>
<th>TAG (mmol/l)</th>
<th>HDL (mmol/l)</th>
<th>LDL (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Mean ± standard deviation) Study subject n = 25</td>
<td>3.14 ± 0.82</td>
<td>1.27 ± 0.37</td>
<td>1.27 ± 0.14</td>
<td>1.30 ± 0.63</td>
</tr>
<tr>
<td>(Mean ± Standard deviation) control subject n = 12</td>
<td>3.08 ± 0.80</td>
<td>1.12 ± 0.44</td>
<td>1.32 ± 0.13</td>
<td>1.21 ± 0.68</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation

When the mean serum Total Cholesterol, High Density Lipoprotein Cholesterol, Low Density Lipoprotein Cholesterol and Triglyceride levels of test subjects were compared with those of the control subjects the difference was not significant (p > 0.05) - (Table 3.1).

When comparison was done within the study group, based on high and normal blood pressure, the mean serum lipid profile level was found to differ significantly with higher mean value from high blood pressure group (p < 0.05) - (Table 3.2).

Table 3.2: Mean ± standard deviation of TC, TG, HDL and LDL among the test subjects with borderline risk of high blood pressure and normal blood pressure.

<table>
<thead>
<tr>
<th>Test Subject</th>
<th>TC (mmol/l)</th>
<th>TAG (mmol/l)</th>
<th>HDL (mmol/l)</th>
<th>LDL (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>borderline risk of high blood pressure</td>
<td>3.74 ± 0.85a</td>
<td>1.44 ± 0.49b</td>
<td>1.20 ± 0.07</td>
<td>1.76 ± 0.91c</td>
</tr>
<tr>
<td>Normal blood pressure</td>
<td>2.0 ± 0.21a</td>
<td>0.95 ± 0.29b</td>
<td>1.35 ± 0.12</td>
<td>0.37 ± 0.21c</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation. Values with similar superscript are significant (p < 0.05)

3.2 DISCUSSION

When the mean serum Total Cholesterol, High Density Lipoprotein Cholesterol, Low Density Lipoprotein Cholesterol and Triglyceride levels of test subjects were compared with those of the control subjects the difference was not significant (p > 0.05) - (Table 3.1). This result is consistent with the findings of previous studies on the relationship between sub-acute or acute sleep deprivation on lipid profile which reported that difficulty falling asleep was not associated with TG or HDL-cholesterol (Troxel et al., 2010). Similarly, a review showed that sleep deprivation does not change total cholesterol levels in rat (Anderson, 2009). Cholesterol
level and Low Density Lipoprotein Cholesterol had no significant change in sub-chronic sleep deprivation (Perry et al., 2007). Research showed that there is no significant changing in cholesterol level in sleep deprived male rats (Antunes, 2007). A study on the effect of sleep deprivation on 18 – 65 years old men and women, proved that short sleep deprivation had no effect on total cholesterol levels (Adamkova, 2009). Thus acute sleep deprivation may be due to acute effect of cardiovascular physiology arising from changes in the autonomic nervous system, rather than Lipid profile (Artham et al., 2008).

However, our study is inconsistent with a report by Dimsdale and Herd that, the level of free fatty acids and total cholesterol increases in chronic stress (Dimsdale and Herd, 1982), which may be attributed to insomnia. Similarly a study conducted by Fakhari and colleagues in 2004 demonstrated increased triglyceride in individuals who had experienced high levels of stress in the preceding 6-12 months (Fakhari et al., 2007). A very recent study has shown that the higher the examination stress the higher the serum Cholesterol, Low Density Lipoprotein Cholesterol and Triglycerides levels and lower the serum High Density Lipoprotein levels, suggesting an increased risk of hyperlipidaemia in stress condition (Uba et al., 2014). However, these reports evaluated effects of chronic sleep deprivation rather than acute or sub-acute sleep deprivation (Pre-insomnia) being reported by our study.

Upon comparison within experimental subjects with borderline risk high blood pressure and normal blood pressure groups, a significant increase (p<0.05)-(Table 3.2) in total cholesterol, triglyceride, low density lipoprotein cholesterol and a decrease (p<0.05)-(Table 3.2) in high density lipoprotein cholesterol levels in individuals with borderline risk of high blood pressure was observed compared with normal blood pressure individuals, suggesting that acute insomnia is associated with prevalence of arterial hypertension. The individuals with border line risk of high blood pressure were thought to be more stressed compared to those with normal blood pressure, indicating an increase in electroencephalogram beta wave activity of the brain, making it harder for the brain to achieve the state of relaxation needed for sleep ( the brain still remain alert ). The body circadian rhythm and time may be disconnected, resulting in disruption of normal sleeping pattern.

Cortisol is a stress hormone secreted at higher level during stress for appropriate stress response. Although the secretion may not be so significant to cause a vast alteration in pre-insomnic individuals, these subjects tend to have an increased blood sugar level thought to be produced by gluconeogenesis through the activation of pyruvate carboxylase, an enzyme
that generate oxaloacetate, an important precursor of glucose in the pathway. The glucose produced provides energy to the brain. This high glucose metabolism leads to the inability of the brain to get full relaxation even during sleep. (Uba et al., 2014). The pre-insomnic condition can thus be attributed to stress as far as our study subjects are concerned.

3.3 CONCLUSION
Our study shows that acute sleep deprivation (pre-insomnia) does not alter serum TC, TG, HDL and LDL cholesterol significantly, however, in stress condition borderline of high blood pressure subjects showed low risk of developing cardiovascular diseases.

4.4 RECOMMENDATION
We recommend further research to investigate cardiac enzymes, blood glucose, cerebrospinal fluid levels of individuals with chronic insomnia.

4.5 ACKNOWLEDGEMENT
We acknowledge the technical assistance of the laboratory technicians, Department of Biochemistry, Bayero University, Kano and S.S Usman for critical reading of the manuscript and making suggestions.

REFERENCE


