

Prolonged Occupational Exposure to Stressful Stimuli(Heat) as a Cause of raised Atherogenic Index in Thermal Power Station Workers

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Abstract :- The aim of this study was to evaluate the relationship between occupational exposure to stressful stimuli (heat) and indicators of hypertension and CHD in workers of the Thermal power station Two Hundred male workers were selected out of whom 100 workers were exposed to heat emitted by boiler in boiler section for 8 hrs daily and 6 days in a week for estimation of lipid profile. The control group consists of office workers and staff who were not exposed to extreme heat. Depending on age, they were divided into four groups. Estimation of lipid profile was carried out by enzymatic method between cases and controls to observe the effect of heat stress on serum lipoproteins like HDL,LDL-C, VLDL-C and LDL/HDL ratio (Atherogenic index). It was observed that there is highly significant increase in serum level of the LDL-C, VLDL-C and non significant changes in HDL-C. Atherogenic index is significantly higher in workers exposed to heat .This suggests that greater is the risk of hypertension and coronary heart disease (CHD) in these workers as compared to controls.

Key words :- Heat stress, LDL, HDL, VLDL, atherogenic index, CHD

INTRODUCTION

Thermal Power station workers may be exposed to various hazards such as heat, metal dusts, fumes, silica, polycyclic aromatic hydrocarbons (PAH), molten metal and machinery. Extremes of temperature have been associated with heart disease, usually in population settings[1] Increased rates of heart disease have been found among occupations exposed to heat, namely potash miners and fire-fighters[1,2]The mechanisms by which heat can lead to cardiac stress, usually in patients with underlying heart disease, are multiple[3]Thermal power station workers working at boiler section may also be at increased risk of cardiac problems due to their exposure to heat which is considered as a stressful stimuli . Psychological stress can lead to heart problems, such as myocardial Infarction[4]and coronary heart disease[5]. Job stress has been shown to be associated with an increased prevalence of cardiovascular diseases among Industrial workers[6]Stressors can be Physical or mental. Examples of physical stressors include exertion, extreme cold, extreme heat, extreme noise, surgery etc.[7] The various physiological changes seen in response to stress are due to increased hypothalamo-pituitary action, activation of pituitary-adrenal system and secretion of various hormones e.g. catecholamines, endorphins and encephalins etc. [8]It is well known fact that stress disturbs the normal physiological equilibrium or homeostasis.[7] Heat stress remains a very important issue even in developed and technologically advanced countries of the world. In industry, such stress may be caused by high ambient temperatures, high humidity, low air movements or high radiant energy sources. Heat is one of the most widespread form of environmental stressor in the industrialized urban areas. . In industry, such stress may be caused by high ambient temperatures, high humidity, low air movements

or high radiant energy sources; each of these may be present on its own or in combination with other factors.[9] Because there is overwhelming evidence the elevated LDL-C concentration in plasma is Atherogenic,[10,11] where as high HDL-C level is cardioprotective[10].The measurement and interpretations of LDL-C and HDL-C levels is emphasized in the US National Cholesterol education program guidelines[12] According to this guidelines, Serum LDL-C should be considered the primary therapeutic target, where as HDL-C levels may also be critical in the assessment of CHD risk. Thus, because serum TG levels are ignored in the National Cholesterol education program algorithm, clinicians are left with LDL-C and HDL-C levels to asses risk while considering the presence or absence of other important risk factors , such as family history of early CHD, age, smoking, hypertension, diabetes mellitus etc. On this basis, LDL/HDL ratio is often calculated to estimate the risk of CHD[12]. The biochemical parameters which has attracted the most sustained and widespread attention in relation to etiology, prevention and treatment of atherosclerosis are serum lipoproteins like LDL,HDL,VLDL and LDL/HDL ratio . Increase levels of serum lipoprotein levels is now recognized as a major risk factors for CHD. Extensive work has been done on the effect of different types of stress such as Examination stress [13] mental stress[14] heat stress [15] in animals and human beings on lipid profile, but there are relatively few studies seeking whether after occupational exposure to heat will have any effect on the serum lipoproteins. The present study intends to focus on occupational exposure to stressful stimuli (heat) on the lipid profile. An Atherogenic index (LDL/HDL ratio) was done with a view to see effect of heat stress on prevalence of CHD in Thermal power station workers.

MATERIAL AND METHOD

SUBJECTS

The subjects were 200 male healthy employees in between age group of 21 -58 yrs from a Thermal power plant that produces electricity. Only males were included in the study as the company employs very less females. The employees work one of three shifts (morning, afternoon or night). The workers were grouped into cases (100) and controls (100). The cases were exposed to heat emitted by boiler in boiler section for 8hours daily and six days a week. Temperature to which they are exposed is in between 360 C to 410C. The control group selected were office workers from the same plant with same age and socioeconomic status. Self-administered questionnaires were used to gather information on demographic characteristics, job history, smoking history (never-, ex-, and current-smoker) and alcohol consumption. All the subjects selected were free from diabetes, cardiovascular or other systemic disorders. Also the smokers and alcoholic were excluded. Depending on age the cases and controls were divided into four groups. (I, II, III & IV)

BIOCHEMICAL ASSAY

The collection of samples of group under study and control has done in cases after the overnight fasting from anticubital vein with all aseptic precautions. The 2ml blood is collected in plain bulb for the estimation of serum lipids and lipoproteins. Blood was allowed to clot at room temperature for half an hour and then centrifuged at 3000rpm for the estimation of lipid profile. After separation of serum, HDL level was estimated by Burstein et al method. [16] LDL-VLDL was estimated quantitatively by Friedwald, Levy and Frederickson method [17] Atherogenic index was calculated by using the formula from (Kayamori and Igarashi, 1994) [18].

STATISTICAL ANALYSIS

The values of serum lipid levels and atherogenic index observed during heat stress were compared to the control values to evaluate their statistical significance by student's t test.[19]

RESULTS

Table no. 1 showed significant decrease in HDL-C in Group III, non significant decrease in group IV while non significant increase in group I & II. A statistically significant decrease in HDL-C was reported in several different stressors by Tsopanakis et al (1984) [20], Singhal S. et al (1997), [8] Solter et al (2002)[2] This findings correlates with our finding in group III. A similar decrease in HDL-C was also observed in group IV however it is statistically non-significant. While significant increase in HDL-C were found by Bijlani et al (1983) [13], stoney et al (1983)[22], Krum et al (1991)[23] In our study, we observed increase in HDL-C levels in group I and II however this increase was statistically non-significant. From the above observation, it is clear that there is no uniformity in the serum levels of HDL-C during stress.

Table-1: Comparision of HDL-C between cases and control belonging to different age group.

| Groups | 21-30yrs | | 31-40yrs | | 41-50yrs | | 51-58yrs | |
|-------------|-----------|-------|------------|-------|-------------|-------|------------|-------|
| | (Group-I) | | (Group-II) | | (Group-III) | | (Group-IV) | |
| | Control | Cases | Control | Cases | Control | Cases | Control | Cases |
| No. | 17 | 16 | 33 | 34 | 38 | 37 | 12 | 13 |
| Mean | | 46.47 | 42.22 | 44.75 | 46.13 | 41.35 | 46.87 | 41.05 |
| | 44.42 | | | | | | | |
| ±SD | 9.19 | 7.26 | 10.31 | 7.78 | 8.97 | 5.16 | 10.95 | 5.68 |
| t- test | P > 0.05 | | P > 0.05 | | P < 0.005 | | P > 0.05 | |
| Significant | N.S | | N.S | | H.S. | | N.S | |

Table No.2. showed a highly significant increase in LDL-C in the age group I, II and IV whereas significant increase in age group III. Our findings of significant increase in LDL-C in all age groups correlates with the studies of Bijlani et al (1983)[13], Tsopanakis et al(1984)[20], Scheuch et al(1991)[24], O' Donnell et al (1993)[25], Mathew et al (1995)[26] and Elizabeth et al (2002)[27].

Table-2 Comparison of LDL-C between cases and control belonging to different age group.

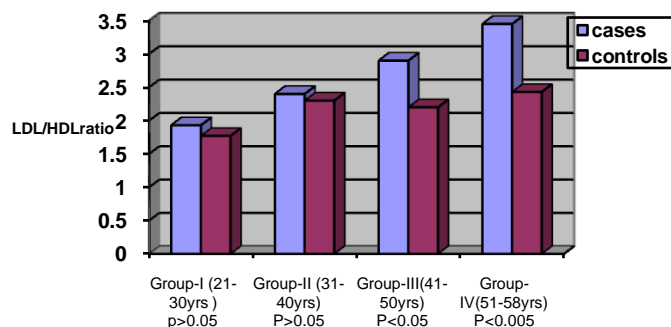
| Groups | 21-30yrs | | 31-40yrs | | 41-50yrs | | 51-58yrs | |
|-------------|-----------|-------|------------|--------|-------------|--------|------------|--------|
| | (Group-I) | | (Group-II) | | (Group-III) | | (Group-IV) | |
| | Control | Cases | Control | Cases | Control | Cases | Control | Cases |
| No. | 17 | 16 | 33 | 34 | 38 | 37 | 12 | 13 |
| Mean | 78.86 | 89.73 | 96.76 | 107.25 | 103.02 | 118.67 | 114.28 | 141.22 |
| ±SD | 12.84 | 10.65 | 28.35 | 19.83 | 26.23 | 29.51 | 27.61 | 15.07 |
| t- test | P < 0.005 | | P < 0.005 | | P < 0.05 | | P < 0.005 | |
| Significant | H.S | | H.S | | H.S. | | H.S | |

From Table no.3, Our study revealed highly significant increase in VLDL-C in group I and III, while significant increase in age group II and IV, which matches with the study reported by Bijlani et al (1983) [13], LeFur C et al (1999) [28] and Hemlata et al (2002) [29]

Table-3: Comparison of VLDL-C between cases and control belonging to different age group.

| Groups | 21-30yrs | | 31-40yrs | | 41-50yrs | | 51-58yrs | |
|-------------|-----------|-------|------------|-------|-------------|-------|------------|-------|
| | (Group-I) | | (Group-II) | | (Group-III) | | (Group-IV) | |
| | Control | Cases | Control | Cases | Control | Cases | Control | Cases |
| No. | 17 | 16 | 33 | 34 | 38 | 37 | 12 | 13 |
| Mean | 12.7 | 17.18 | 14.22 | 17.75 | 19.58 | 24.09 | 22.81 | 27.67 |
| ±SD | 3.2 | 4.22 | 4.44 | 6.36 | 6.56 | 5.97 | 3.5 | 8 |
| t- test | P < 0.005 | | P < 0.01 | | P < 0.005 | | P < 0.05 | |
| Significant | H.S | | H.S | | H.S | | H.S. | |

Fig. 1 LDL/HDL ratio (Atherogenic Index) between cases and controls belonging to different age groups.



P < 0.005 → highly significant,

P < 0.05 → significant,

P > 0.05 → non-significant

Figure No. 1 shows highly significant increase in Group IV, significant increase in Group III, and non-significant increase in Group I and II.

DISCUSSION

The present study indicates an influence of prolonged exposure to heat on the Serum lipoproteins. In the present study, it is clear that there is no uniformity in the serum levels of HDL-C during stress. Sangeeta singhal et al (1997)[8] observed significant decrease in HDL-C in immobilization stress and proposed an hypothesis that this decline is due to increase steroidogenesis in adrenals for which HDL-C is mobilized to adrenals during stress. Hemlata et al (2002) [29] observed an inverse relation between lipid peroxidation and HDL-C levels. As during stress, lipid peroxidation is increased, it may be responsible for decrease HDL-C levels. Mathew et al (1995) [26] and Krum et al (1999) [23] observed increase HDL-C levels during stress and strongly claimed that it is due to haemoconcentration. Bijlani et al (1986) [13] claimed that even though the reason for this discrepancy in HDL-C is not clear but it could reflect genetic differences. Our study showed significant increase in LDL-C and VLDL-C. The increase in LDL-C and VLDL-C levels after exposure to stress might be due to:

- ◆ It is observed that during periods of stress, there are alterations in the level of serum cholesterol, triglycerides, LDL-C and VLDL-C to meet the extra metabolic demands of body tissues. Thus, during stressful periods, there is increase in the blood levels of a number of hormones like cortisol, epinephrine, nor-epinephrine and growth hormone which are lipolytic in nature and they mobilize the lipid stores of adipose tissue and Liver to meet the extra-caloric requirement of tissue.[30]
- ◆ Mathew et al in (1995)[26] in their work on psychological stress proposed that the cardio vascular responses to stress sufficiently increase hydrostatic pressure to cause a net movement of fluid out of the intra vascular space and consequently, concentrate all non diffusible blood constituents. Thus, elevation of LDL-C and VLDL-C during stress was caused by accompanying haemoconcentration. However in heat stress the haemoconcentration is further accentuated by the water loss by sweat[31]
- ◆ Sympathetic activity is increased during stress. Stimulation of the sympathetic innervation to fat releases nor-epinephrine, which acts via β_3 adrenergic receptors to increase lipolysis[30, 32]
- ◆ However, Bijlani et al in (1996) [13] suggested that this rise in LDL-C and VLDL-C is essential for increased synthesis of ACTH and glucocorticoids during stress for which it acts as a precursor.

Our study revealed some differences in LDL/HDL ratio (Atherogenic index) when compared between case and control. present study showed significant increase in LDL/HDL ratio only in Group II and IV which might be due to age and duration of exposure to heat. A significant increase of atherogenic index was observed, primarily due to the decreased serum HDL C levels, suggesting these patients are possibly exposed to higher risk of atherosclerosis. All these were associated with higher risk for development of CHD.

CONCLUSION

Thus we observed a significant increase in serum lipoproteins and atherogenic index in workers of thermal power station those who are exposed to heat when compared with controls. This suggests that the workers are at higher risk of hypertension and CHD which is also dependent on age and duration of exposure.

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