

CIRCULATING LEUKOCYTES; RELATION WITH ACUTE MENTAL STRESS & CORONARY ARTERY DISEASE

DR. ISRAR AHMED AKHUND

MBBS, M.Phil

Professor of Physiology
Muhammad Medical College,
Mirpurkhas

DR. IRSHAD ALI ALVI

MBBS, FCPS

Assistant Professor of Medicine
National/Peoples Medical College,
Nawabshah

DR. GHULAM RASOOL BHURGRI

MBBS, M.Phil

Assistant Professor of Pharmacology
Muhammad Medical College,
Mirpurkhas

Dr. Muhammad Ali Qureshi, MBBS

Demonstrator of Anatomy
Muhammad Medical College,
Mirpurkhas

Dr. Haji Khan Khoharo

MBBS, FCPS

Assistant Professor of Medicine
Muhammad Medical College Hospital,
Mirpurkhas

Article Citation:

Akhund IA, Alvi IA, Bhurgri GR, Qureshi MA, Khoharo HK. Circulating leukocytes; Relation with acute mental stress & coronary artery disease. Professional Med J Sep 2010;17(3):455-458.

ABSTRACT... Objective: Evaluating circulating leukocytes in acute mental stress & relation with coronary artery disease. **Design:** Descriptive study **Setting:** Muhammad Medical College Mirpurkhas, **Duration:** from March 2007 to August 2007. **Methods:** Two hundred young healthy adults were studied for stress experiment. Venous blood samples were drawn before and after stress for estimation of leukocyte counts. Values were presented as mean \pm standard error of mean (SEM). **Results:** The difference in Pre and during stress results of variables were TLC = -4630.85 \pm 140.65, N % = -11.8 \pm 0.36, L% = 4.03 \pm 0.14, M % = 5.48 \pm 0.37, E % = 1.18 \pm 0.07, B % = 1.11 \pm 0.022. Highly significant p-values (\leq 0.001) were found among various parameters, in both groups of subjects. **Conclusion:** An increase in the number of circulating leukocytes was an important unexpected observation that was noted. We suggest that the real life stress induced leukocytes changes may warrant further investigation about its relation with the coronary artery disease (CAD).

Key words: Mental Stress Leucocytes Coronary Artery Disease.

INTRODUCTION

Considerable progress has been made towards identifying personal and environmental characteristics that predispose to or predict the occurrence of coronary artery disease (CAD)¹. The risk factors that have been confirmed include increased positive family history, male gender, age, hypertension, hypercholesterolemia, diabetes mellitus, cigarette smoking, stress, physical inactivity, obesity and genetic prediposition²⁻⁴.

However, there is still much to be learned about the

prediction of CAD. The frequency of CAD in different parts of the world has been found to be related to the mode of life⁵. Consequently, in recent years many investigators have sought to unravel the complex relationship between

Article received on: 30/09/2008
Accepted for Publication: 15/10/2009
Received after proof reading: 04/08/2010
Correspondence Address:
Dr. Haji Khan Khoharo
B. No. C-17/II, Anwer Villas, Phase-I
New Wahdat Colony Qasimabad
Hyderabad-Sindh
drhajikhan786@yahoo.com

the incidence of CAD and emotional stress. Extensive evidence suggests that the most characteristic trait of the young coronary victim is his emotional stress⁶.

At present there is a clear understanding of the manner in which emotional stress may lead to CAD. A recent study suggests that the stress induced circulating catecholamine's can increase the leukocytes count⁷. The leukocytosis has been consistently shown to be an independent risk factor and prognostic indicator of future cardiovascular outcomes, regardless of disease status. The leukocyte count is inexpensive, reliable, easy to interpret, and ordered routinely in inpatient and outpatient settings⁷. In order to confirm this fact, we have carried out this study by using extensive data from young healthy adults of this well known medical college of Sindh province.

SUBJECTS & METHODS

Two hundred young healthy adults were studied. Informed consent was obtained from all the subjects before their entry. Stress experiments were performed

according to highly standardized procedure⁸. Subjects were instructed to avoid heavy exercise and to be fasting for two hours before examination. About 2 ml of venous blood samples were drawn from antecubital vein under aseptic conditions, during their class test examination (as stress condition) and after their test examination during pres-stress condition (controls), for estimation of leukocytes counts, which were determined according to Dacie and Lewis⁹. Standard statistical methods were used. Values were presented as mean \pm standard error of mean (SEM). Differences between means were evaluated by Students t-test for paired comparisons¹⁰.

RESULTS

When results were summed up and test parameters were compared, it was seen that (Table) the difference in Pre and During stress results of variables were TLC = -4630.85 \pm 140.65, N % = -11.8 \pm 0.36, L% = 4.03 \pm 0.14, M % = 5.48 \pm 0.37, E % = 1.18 \pm 0.07, B % = 1.11 \pm 0.022. When compared statistically, the differences were found highly significant (p-value \leq 0.001) among various parameters in both groups of subjects (Table-I).

Table-I. Leukocyte variables, the paired "t" test with p-value significance.

| | TLC(: L) | N% | L% | M% | E% | B% |
|------------------|--------------|--------------|--------------|--------------|--------------|--------------|
| Pre-stress | 7218.15 | 57.2750 | 7.2000 | 32.4850 | 1.9300 | 1.1100 |
| During stress | 11849 | 69.0750 | 3.1700 | 27.0050 | .7500 | .0000 |
| Mean difference* | -4630.85 | -11.8 | 4.03 | 5.48 | 1.18 | 1.11 |
| P-value | \leq 0.001 | \leq 0.001 | \leq 0.001 | \leq 0.001 | \leq 0.001 | \leq 0.001 |

*Mean differences of pre-stress minus during stress.

DISCUSSION

Mental stress, acting through cerebral cortex, causes physiological stimulus which is similar to the defense alarm mechanism. The changes may be contributed through neurohormonal and hormonal mechanisms, which cause initiation and progression of physiological changes. Present study has shown by a rise in the leukocytes during acute mental stress, which is in agreement with the previous reports^{7,11-13}. Our results are similar to that of a recent study done by Jern et al, who

conducted the study on twenty healthy adults⁶. We found a significant increase in leukocyte count during class test examination. The relative increments were significantly greater. This is perhaps because of elevated catecholamines levels during this condition. Another clinical study done by Jern et al suggests that persons with higher leukocyte counts have greater risk of myocardial infarction. They concluded that this increase in risk of myocardial infarction associated with increased blood leukocytes was similar to that found for cholesterol

and blood pressure¹³. Therefore, our results approach to be an important clinical suggestion that total leukocyte counts may be useful in screening the risk of CAD, since the leukocyte count is so frequently detrimental and so economical to measure that it may be helpful in assessment of CAD, as it seems to provide an additional information; i.e., the leukocytes may possess an additional predictive power as indicated by Unern et al. Another recent study has demonstrated strong relationship between circulating monocyte-platelet aggregates (MPA) and acute myocardial infarction. This study demonstrated that MPA may appear in the circulation earlier than routine markers of myocardial necrosis such as CK-MB. Thus MPA may be a marker of plaque instability and intracoronary dynamics of thrombosis and inflammation¹⁴.

Another study has shown the leukocyte count as a risk factor for coronary artery bypass graft mortality¹⁵. Another more recent study has shown the high leukocyte count was associated with increased CAD morbidity and mortality in various patients' population and clinical settings. It also appears to be an independent risk factor, regardless of atherosclerotic disease status⁷. Therefore, present study warrants about this relation to conduct further studies, with specific attention to all components of the circulating leukocytes, which may reflect this important pathophysiological association between leukocytes and CAD, which is still unclear.

CONCLUSION

An increase in the number of circulating leukocytes was an important unexpected observation that was noted. We suggest that the real life stress induced leukocytes changes may warrant further investigation about its relation with the coronary artery disease (CAD).

Copyright© 15 Oct, 2009.

REFERENCES

- Schildkraut JM et al. **Coronary risk associated with age and sex of parental heart disease in Framingham study.** Am J Cardiol 1989;64:555.
- Bashore TM, Granger CB, Hranitzky P. In: Tierney LM Jr, McPhee SJ, Papadakis MA, editors. **Current medical diagnosis and treatment.** 47th ed. New York: Mc Graw-Hill; 2008:300-303.
- Boon NA, Fox KAA, Bloomfield P, Bradbury A. **Cardiovascular disease.** In: Haslett C, Chilvers ER, Boon AB, Colledge NR, Hunter JAA, editors. Davidson's principles and practice of medicine. 19th ed. Churchill Livingstone: Elsevier sciences limited;2002:422-23.
- Camm AJ. **Cardiovascular disease.** In: Parveen Kumar, Michael Clark, editors. Clinical medicine. 5th ed. W. B Saunders: Elsevier sciences limited UK;2002:767-69.
- Carlenton RA, Dwyer J et al. **Report of the expert panel on population strategies for blood cholesterol reduction.** Nat Inst Health Cir 1991;83:2154-2232.
- Jern S. **Psychological, hematological and hemodynamic factors in borderline hypertension.** Acta Medica Scand 1982:662.
- Majid M, Awan I, Willerson JT, Casscells SW. **leukocyte count and coronary heart disease.** J Am Coll Cardiol, 2004;44:1945-56,doi: 10.1016/j.jacc.2004.07.056.
- Dacie SJV, Lewis SM. Practical hematology Sir John Dacie and S.M Lewis 6th edi. Edinburgh; Churchill Livingstone 1984:8.
- Sgoutas SA, Cacoppa JT, Uchimo BN and Malakay W: **the effects of an acute psychological stressor on cardiovascular, endocrine and cellular response.** Psychophysiology 1994;31(3):264-71.
- Armitage P, Berry G. **Statistical methods in medical research.** Oxford: Blackwell Scientific Publications 1998:456-66.
- French EB, Steel CM, Aitichison WR. **Studies on adrenaline induced leukocytosis in normal man.** Br J Haem 1971;21:423-28.
- Patterson SM, Matthew KA et al. **Stress induced haemoconcentration of blood cells and lipids in healthy women during acute mental stress.** Health Psychiatry 1995;14:318-23.
- Friedmann GD, Klatsky AL, Siegelaub AB. **The leukocyte count as predictor of myocardial infarction.** N Eng Med J 1974;290:1275-8.
- Furman MI, Bernard MR, Lori A, Krueger BA, Marsha L, Fox RN, et al. **Circulating monocyte-platelet**

aggregates are an early marker of acute myocardial infarction. *J Am Coll Cardiol*, 2001;38:1002-6.

factor for coronary artery bypasses graft mortality. *Am J Med*, 2003;115(8):660-3, doi: 10.1016/S 0002-9343(03)00438-8.

15. Bagger JP, Zindrou D, Taylor KM. leukocyte count: a risk

sta in hoc volumine.

Epibosini et abt. zibosini.
Epibosini Damasceni.
Liber secretorum zibosini.
Liber pnothocantoni.
aspectu planetarum.
Liber qui dicitur zibosini.
Liber de elementis.
zibosini.
Liber de aere.
Liber de igne.
Liber de terra.
Liber de aqua.

H-2000

INDEPENDENT REVIEWS

A soul mate in writing

Vol:12, No.1-3
2010
Jan, Feb, Mar

www.indepreview.com