

Estimation Serum Homocysteine Level in Coronary Artery Disease Patients**Gaurav Singhal¹, Dinesh Gautam², Sohan K. Sharma³, Shyam Sunder⁴**¹Associate Professor and HOD, Department of Cardiology, ESIC Medical College and Hospital, Jaipur, Rajasthan²Professor, Department of Cardiology SMS Medical College, Jaipur, Rajasthan³Professor, Department of Cardiology, SMS Medical College, Jaipur, Rajasthan⁴Professor and Head, ESIC Medical College and Hospital, Jaipur, Rajasthan

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Abstract:

Globally, coronary artery disease (CAD) has become a major public health issue and main contributor to mortality and morbidity and its prevention and effective treatment modalities are key strategies in reducing the mortality. India is in epidemiological transition. To estimation serum homocysteine level in coronary artery disease patients and normal controls. This descriptive study was conducted on 30 patients with confirmed CAD and 30 healthy age matched subjects as controls. In this study, serum homocysteine ($34.16 \pm 15.32 \mu\text{mol/l}$) levels were significantly elevated in CAD patients compared to controls. Lipid profile parameters such as serum cholesterol ($210.39 \pm 41.23 \text{mg/dl}$), TGL ($210.36 \pm 53.26 \text{mg/dl}$), LDL ($131.02 \pm 44.0 \text{mg/dl}$), VLDL ($43.02 \pm 9.12 \text{mg/dl}$) were significantly increased and HDL ($37.12 \pm 9.12 \text{mg/dl}$) levels were decreased in CAD patients compared with healthy subjects. This study show that the serum homocysteine level are significantly elevated in CAD patients compare to controls.

Keywords: Coronary artery disease [CAD], Homocysteine, LDL, VLDL, HDL.

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Introduction

Coronary artery disease (CAD) remains one of the leading causes of morbidity and mortality worldwide. It is a multifactorial disorder influenced by both conventional risk factors such as hypertension, diabetes mellitus, dyslipidemia, smoking, obesity, and sedentary lifestyle, as well as emerging biochemical markers that may play an important role in its pathogenesis. [1] According to the World Heart Federation, 35 % of all CVD deaths in India occur in those aged 35–64 years. 90–95 % of all cases and deaths are due to CAD. Approximately, one-sixth of world's population lives in India and CAD remains the highest cause of mortality in India. [2]

Atherosclerosis is characterized by a thickening of the arterial wall due to smooth muscle cell proliferation, lipid deposits, and fibrosis. Rupture of the lipid-containing atherosclerotic plaques results in thrombosis (atherothrombosis) and leads to myocardial infarction (MI) and stroke. [3,4] Therefore, CAD is the narrowing or blockage of the arteries and vessels that supply oxygen and nutrients to heart. MI is one of the manifestations of CAD. MI is a disease of the vessel that feeds the cardiac muscle, called the coronary artery.

Several epidemiological and clinical studies have demonstrated that hyperhomocysteinemia is an independent risk factor for CAD, comparable to traditional factors. The mechanisms proposed include impairment of nitric oxide bioavailability, promotion of low-density lipoprotein oxidation, and direct toxic effects on vascular endothelium. Genetic polymorphisms, nutritional deficiencies of folate, vitamin B6, and vitamin B12, as well as renal dysfunction, are important determinants of serum homocysteine levels. [5-6]

Given the global burden of CAD and the need to identify modifiable risk factors, estimation of serum homocysteine levels may help in early detection of individuals at high risk, guide preventive strategies, and contribute to better clinical outcomes. Therefore, the present study is undertaken to estimate serum homocysteine levels in patients with coronary artery disease and to assess its possible role as a biomarker in cardiovascular risk stratification.

Methodology**Type of study:** Descriptive cross-sectional study**Including Criteria:** 30 patients suffering from CAD as study group & age sex matched 30 normal

controls. The CAD diagnosis was based on clinical history (angina pain), ECG findings, elevated cardiac markers.

Exclusion Criteria: The patients with renal disease, liver disease, diabetes mellitus, respiratory failure and those on drugs influencing the homocysteine level were excluded from this study.

Method of data collection: Under aseptic conditions, 5 ml fasting venous blood samples were collected, centrifuged at 3000 rpm for 10 minutes. The obtained serum sample was used for the estimation of total cholesterol, triglycerides, HDLC

were done by using commercially available autoanalyzer kits and homocysteine by ELISA method. LDLC and VLDL were calculated. Demographic details were collected from the subjects.

Statistical analysis: The results were expressed in Mean \pm SD. Mann-Whitney U test was used for the comparison of non-normally distributed variables. P value <0.05 consider as significant.

Result

Table 1: Socio-demographic profile

Variable	Case	Control	p-value
Age in years	46.11 \pm 9.17	45.6 \pm 8.19	>0.05
Male : Female	16 : 14	15 : 15	>0.05

The mean age of CAD patients was 46.11 \pm 9.17 years and in controls was 45.6 \pm 8.19 years. Both groups were comparable.

Table 2: Biochemical parameters

Variable	Case	Control	p-value
Serum homocysteine (μ mol/l)	34.16 \pm 15.32	9.02 \pm 3.08	0.001
Serum Cholesterol (mg/dl)	210.39 \pm 41.23	156.32 \pm 29.35	0.001
Serum triglycerides (mg/dl)	210.36 \pm 53.26	138.10 \pm 67.01	0.001
Serum HDL (mg/dl)	37.12 \pm 9.12	40.12 \pm 11.0	0.24
Serum LDL (mg/dl)	131.02 \pm 44.0	96.12 \pm 39.02	0.001
Serum VLDL (mg/dl)	43.02 \pm 9.12	31.06 \pm 5.13	0.001

In this study, serum homocysteine (34.16 \pm 15.32 μ mol/l) levels were significantly elevated in CAD patients compared to controls. Lipid profile parameters such as serum cholesterol (210.39 \pm 41.23mg/dl), TGL (210.36 \pm 53.26 mg/dl), LDL (131.02 \pm 44.0 mg/dl), VLDL (43.02 \pm 9.12 mg/dl) were significantly increased and HDL (37.12 \pm 9.12 mg/dl) levels were decreased in CAD patients compared with healthy subjects

Discussion

The present study shows significantly increased serum homocysteine level in CAD patients as compared to control subjects. The mean serum homocysteine level was found to be increased to (34.16 \pm 15.32 μ mol/l) with a range of 10-14 μ mol/l in patients of CAD. The results were in close conformity with the findings of Yadav et al [7] and Tahir et al [8]. The mean serum homocysteine level was observed to be 9.19 \pm 3.06 μ mol/l with a range of 5 -15 μ mol/l in normal control subjects (table no.1).The results were in close collaboration with the observation made by Jaffrey et al [9]. Since 1992 there are several studies indicating that elevated homocysteine was an independent graded risk factor for atherosclerotic disease in coronary, cerebral and peripheral arteries [10-12] Lagrand et al [13] determined that serum C-RP level is very highly significant(p<0.001) when compared to control

subjects. Aaron et al [14] determined the elevated serum C-RP concentration in atherosclerotic patients. Beamer et al [15] have also reported that stroke patients without infection have increased level of C-RP.

Conclusion

The present study concludes that significantly elevated homocysteine and dyslipidaemia in CAD patients compared to healthy controls, suggesting that homocysteine and traditional cardiovascular risk factors may be synergistically prompt the formation and development of atherosclerosis in CAD patients. Further studies with large sample size are recommended.

References

1. Davies MJ. Stability and instability: two faces of coronary atherosclerosis. The Paul Dudley White Lecture 1995. Circulation 1996; 94: 2013–20.
2. Mudd SH, Skovby F, Levy HL, Pettigrew KD, Wilcken B, Pyeritz, RE et al. The natural history of homocystinuria due to cystathionine beta-synthase deficiency. Am J Hum Genet 1985; 37:1–31.
3. Shwan Othman Amen, Soza Tharwat Baban. Association of Hyperhomocysteinemia with Acute Myocardial Infarction in Kurdish

- Patients. IOP Conf. Series: Materials Science and Engineering.2019; 557:1-10.
4. Wilson PW. Assessing coronary heart disease risk with traditional and noel risk factors. Clin Cardiol 2004; 27: III7-11.
 5. Venes D, Clarence WT. In: Venes D, editor. Taber's Cyclopedic Medical Dictionary. 21st ed. Philadelphia: F.A. Davis; 2005. p. 1089.
 6. Hankey GJ, Eikelboom JW. Homocysteine and vascular disease. Lancet. 1999; 354:407-13
 7. Yadav AS., Bhagwat VR., and Rathod IM.: "Relationship by plasma homocysteine with lipid profile parameters in ischemic heart disease." I.J.C.B. 21 (1): 106-110; 2006.
 8. Tahir Y., Maral G., Ahmet G., Fulya I and Ednan B.: The serum high sensitive C-reactive protien and homocysteine levels to evaluate the prognosis of acute ischemic stroke dept. of cardiology, Turkey: 145-151, 2007.
 9. Jeffrey A.C., Robert W.E., Shaten B.J., John D.H., Lewis H. K: Homocysteine and risk of cardiovascular disease in the multiple risk factor intervention trial. I.H.J.; Nov-Dec (suppl) S44-S52. 2000.
 10. Selhub J, D'Angelo A.: Homocysteine and thrombotic disease. Blood; 90, 1-11, 1997.
 11. Boushey C.J., Beresford S.A.A., Omenn G.S. et al.: A quantitative assessment of plasma homocysteine as a risk factor for vascular disease: Probable benefits of increased folic acid intakes. J.A.M.A.; 274, 1049-1057, 1995.
 12. Ghambhir D.S.: Homocysteinemia and risk for cardiovascular disease. Incl. Heart J. Nov-Dec (suppl), 2000.
 13. Lagrand W.K. et al.: C-reactive protein as a cardiovascular risk factor. More than an epiphenomenona. Circulation; 100, 96-102, 1999.
 14. Aaron R. Folsom. et al.: Association of C-reactive protein with marker of prevalent atherosclerotic disease. Am. J. Cardio.; 88, 1 12-117, 2001.
 15. Beamer, NB., Coull. B.M., Clar, W.M., Hazel, S. and Silberger, J.R.: "Interleukin-6 and interleukin-1 receptor antagonist in acute stroke." Annals of Neurology. Vol. 37, 800-805, 1995.