

Hyperhomocysteinaemia and Acute Myocardial Infarction in Patients Admitted at Isra University Hospital, Hyderabad

IKRAM DIN UJJAN, IMRAN SHEIKH, ASIF ALI BURNEY, ASIM JAMAL SHEIKH, NAHEED PARVEEN, RASHID AHMED MEMON, ABDUL RAHIM MEMON, MUHAMMAD FAROOQ,, MUHAMMAD UZAIR SAQLAIN.

ABSTRACT

Myocardial infarction is a major consequence of coronary artery disease. Recently many reports have been suggested that hyperhomocysteinemia had an important role in myocardial infarction. Plasma homocysteine level was determined in 60 myocardial infarction patient (Group A) and 35 healthy controls (Group B). Statistically significant difference was observed in plasma homocysteine concentrations between the patients of acute myocardial infarction (Group A) and in normal healthy individuals (Group B). The level of homocystine in patients of myocardial infarction is significantly increased when compared with controls. This indicates a strong association between hyperhomocysteinemia and acute myocardial infarction in the peoples of Hyderabad, thus showing plasma homocysteine as a risk factor for myocardial infarction.

Key Words: Myocardial Infarction, Homocysteine

INTRODUCTION

Acute myocardial infarction is a clinical syndrome that results from an injury to myocardial tissue that is caused by an imbalance between oxygen supply and demand. Hypertension, arteriosclerosis, smoking and alcohol consumption are the risk factors, causing acute myocardial infarction¹.

Abnormal homocysteine levels appear to contribute to coronary artery disease by the following mechanisms: (a) causing endothelial dysfunction. It is an early marker of atherosclerosis, (b) causing marked impairment of endothelium-dependent vasodilatation in response to acetylcholine and adenosine diphosphate (ADP); (c) causing oxidative stress on endothelial cells which is mediated by hydrogen peroxide, (d) promoting the growth and proliferation of vascular smooth muscle, (e) decreasing the homocysteine, (f) a direct toxic effect that damages the cells lining inside the arteries; Elevated homocysteine may cause cardiovascular disease by a reduction in plasma or tissue adenosine levels. Adenosine has wide variety of protective effects on cardiovascular homeostasis. It causes coronary and cerebral artery vasodilatation, increases blood flow in the microcirculation, inhibits platelet aggregation and decreases proliferation or growth of smooth muscle or mesangial cells^{3,4}.

*Pathology Department, ISRA University Hyderabad.
Correspondence to Dr. Ikram Din Ujjan*

METHODOLOGY

Sixty patients, admitted in coronary care unit in ISRA University Hospital Hyderabad, were included in this study. Fasting blood sample was collected from 60 patients (Group A) and 35 healthy subjects (Group B). 3ml sample was taken in EDTA coated tubes & plasma was separated. Homocystine level was measured, by using commercially available kit. The value was considered normal when below 15µmol/L. Students "t"-test was used to compare the results.

RESULTS

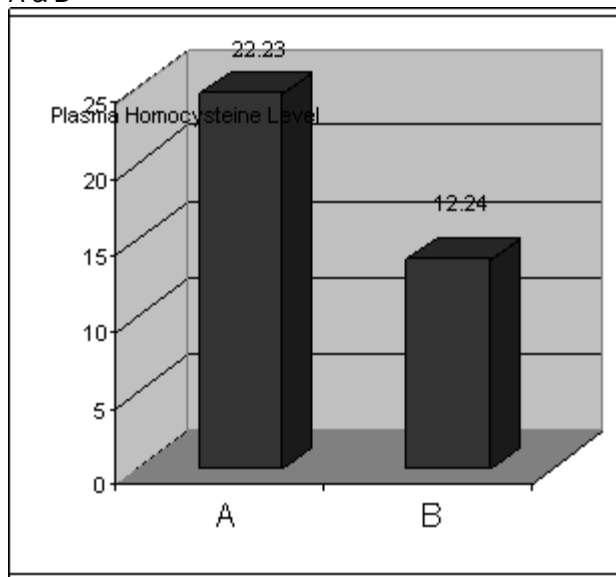
Regarding homocysteine levels, there is an elevation of plasma homocysteine level in patients of acute myocardial infarction (Group A). The difference is highly significant when compared with control group (Group B) (Table 1). Male: Female ratio in group A and B subjects was 4.5: 1 & 4:1 respectively.

Table 1: Comparison of plasma homocysteine level in group A & B

Plasma Homocysteine Level (µmol/L)	Group A (patients with Acute MI)	Group B (Healthy subjects)
Mean±SD Value	24.59± 3.54	12.24± 2.12
Total Subjects	60	35

Statistical Analysis: A Vs B= P < 0.01 (Highly Significant)

Fig 1: Comparison of plasma homocysteine level in group A & B



DISCUSSION

There is an elevation of plasma homocysteine in patients of acute myocardial infarction. Statistically significant difference ($P < 0.01$) was observed when comparing group A (patients with acute MI) and group B (control) subjects (Table 1). Recent reports on homocysteine suggest that it is an independent predictor of vascular disease, including stroke and coronary artery disease. Mutations in the methylenetetrahydrofolate reductase gene is one of the most frequent causes of moderately elevated plasma homocysteine. A meta analysis conducted by Boushey et al (1995)⁵, showed that homocysteine was an independent risk factor for atherosclerosis in the coronary, cerebral and peripheral vessels. Plasma homocysteine concentration is found to be higher in Asians compared to the North American and European whites. A study conducted by Stampfer et al (1992)⁶ have concluded that moderately high levels of plasma homocysteine are associated with subsequent risk of myocardial infarction. Christophe et al (2000)⁷ have reported that elevated plasma homocysteine concentration is a risk factor for

coronary heart disease, independent of conventional risk factors.

Homocysteine is known to induce atherothrombosis in many ways: homocysteine thiolactate, a by product of oxidation of homocysteine combines with LDL to form foam cells. The LDL rich foam cells embed themselves in the vascular endothelium and become fatty streak, which is the beginning of an atherosclerotic plaque^{8,9}.

REFERENCES

1. Loehrer FMT, Angst CP, Haefeli WE, Jordan PP, Ritz R, Fowler B: Low whole-blood S-Adenosylmethionine and correlation between 5-methyltetrahydrofolate and homocysteine in coronary artery disease. *Arterioscler Thromb Vasc Biol* 1996; 6: 727-733.
2. Evans RW, Shaten J, Hempel JD, Cutler JA, Kuller LH: Homocyst(e)ine and risk of cardiovascular disease in the Multiple Risk Factor Intervention Trial. *Arterioscler Thromb Vasc Biol* 1997; 7: 1947-1953.
3. Nygard O, Nordrehaug JE, Refsum H, Ueland PM, Farstad M, Vollset SE. Plasma homocysteine levels and mortality in patients with coronary artery disease. *N Engl J Med* 1997; 337: 230-236.
4. Hofmann MA, Amiral J, Kohl B, Fiehn W, Zumbach MS, Ziegler R et al. Hyperhomocyst(e)inemia and endothelial dysfunction in IDDM. *Diabetes Care* 1998; 20: 1880-1886.
5. Boushey CJ, Beresford SA, Omenn GS, Motulsky AG. A quantitative assessment of plasma homocysteine as a risk factor for vascular disease: probable benefits of increasing folic acid intakes. *JAMA* 1995; 274: 1049-1057.
6. Stampfer MJ, Malinow MR, Willett WC. A prospective study of plasma homocyst(e)ine and risk of myocardial infarction in US physicians. *JAMA* 1992; 268: 877-881.
7. Christophe MT, Michele PC, Jean PML. Plasma Homocysteine and Severity of Thoracic Aortic Atherosclerosis; Institution: Beijing Book Company, Chest. 2000; 118: 1685-1689.
8. Stamler JS, Osborne JA, Jaraki O. Adverse vascular effects of homocysteine are modulated by endothelium derived relaxing factor and related oxides of nitrogen. *J Clin Invest* 1993; 91: 308-18.
9. Durand P, Lussier-CS, Blache D. Acute methionine load- induced hyperhomocysteinaemia enhances platelet aggregation thromboxane biosynthesis, and macrophage derived tissue factor activity in rats. *FASEB J* 1997; 11: 1157 – 68.