MYOCARDIAL INFARCTION

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ABSTRACT... Objective: To assess the frequency of hyperhomocysteinemia in patients of first attack of ST-segment elevated myocardial infarction under age of 40 years. **Design:** Cross sectional study. **Period:** December 2009 to June 2010. **Setting:** Ch. Pervaiz Elahi Institute of Cardiology, Multan. **Material and methods:** A total of 65 patients of acute myocardial infarction were included in the study on the basis of chest pain, ECG changes and increased cardiac enzyme. **Results:** A total of 65 patients fulfilled the inclusion criteria were included the study. Mean age was 35.68 years. Out of the 65 patients, 87.7% were male and 12.3% were female, 41.5% patients has increased homocysteine level while 68.5% had normal homocysteine level. **Conclusions:** Plasma Hcy is an important risk factor for the development of the acute myocardial infarction.

Key words: Homocysteinemia, Acute myocardial infarction.

INTRODUCTION

Acute myocardial infarction (AMI) is one of the most common, serious, chronic and life threatening illnesses in the world. In United States, more than 12 million persons have ischemic heart disease (IHD), more than 6 million have angina pectoris and more than 7 million have sustained AMI¹. In this country, approximately 650,000 patients experience a new AMI and 450,000 experience a recurrent AMI each year. As we know the main pathophysiologic basis for IHD and MI is coronary artery disease (CAD) that causes reduction in oxygen supply to the cardiac tissue. On the other hand the main cause of the CAD is atherosclerosis. Different factor like high plasma level of low density lipoprotein (LDL), low plasma level of high density lipoprotein (HDL), cigarette smoking, hypertension, diabetes mellitus and hyperhomocysteinemia are the major culprits for atherosclerosis. However, a large number of CAD patients do not have any of these known risk factors; therefore, the cause of atherosclerotic CAD in these patients is difficult to explain².

Association of atherosclerosis and CAD has been reported with a few infectious agents such as gram negative bacteria, chalmpydia pneumonia, herpes viruses and helicobacter pylori. Acute myocardial infarction and unstable angina are part of a spectrum known as the acute coronary syndromes, which have in common a ruptured atheromatous plaque³. Plaque rupture results in platelet activation, adhesion and aggregation, leading to partial or total occlusion of the artery. Acute coronary syndromes include STsegment elevation MI, non-ST segment elevation MI and unstable angina.

In the past decade, the number of people who die each year of MI has decreased significantly. Both in hospital mortality and out of hospital mortality have declined as a result of substantial increases in the uses of aspirin, heparin, thrombolytic therapy and coronary angioplasty as well as reduction in the risk factors for CAD e.g. hypertension, hyperlipidemia, smoking and sedentary lifestyle. However, it must be emphasized that there is unfortunately, persistent discordance between existing guidelines for management of AMI and current clinical practice.

This study was carried to assess the frequency of hyperhomocysteinemia in patients of first attack of ST-segment elevated myocardial infarction under age of 40 years.

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MATERIAL AND METHODS

This cross sectional study was carried out from December 2009 to June 2010 at Ch. Pervaiz Elahi Institute of Cardiology, Multan. A total of 65 patients of acute myocardial infarction were included in the study on the basis of chest pain, ECG changes and increased cardiac enzyme.

RESULTS

Out of 65 patients, 57 (87.7%) were male and 8 (12.3%) were female (Table-I).

Table-I. Sex distribution				
Sex	No. of patients	%age		
Male	57	87.7		
Female	08	12.3		

Mean age of the patients was 35.68 years and median age was 35 years, SD was 2.895, range of the age was 14 years (Table-II).

Table-II. Mean age of patients in years				
Total no. of patients	65			
Mean age	35.68			
Median age	35.00			
Mode age	35			
Std. Deviation	2.895			
Range	14			
Minimum age	25			
Maximum age	39			

Out of male patients 40.4% had increased homocysteine level while 59.6% had normal homocysteine level. When we saw the ratio of hyperhomocysteine level, we found that 50% of female population had hyperhomocysteinemia while 50% had normal level of homocysteine level (Table-III).

Hyperhomocysteinemia was also different in different age groups. In 21-30 years of age group, only 1 patient had hyperhomo-cysteinemia while 4 patients had normal level of homocysteine (Table-IV).

Presentation of chest pain could be assessed in the patients of different age groups. We could find out that in 21-30 years of age group, it was evident that 4 patients had chest pain while 1 patient had no chest pain. While in 31-40 years age group, it was clear that 57 patients had chest pain and 3 patients had no chest pain as shown in table-V.

Table-III. Hyperhomocysteinemia in different gender groups				
Sex	Hyperhomocysteinemia			
	Yes	No		
Male	23 (40.4%)	34 (59.6%)		
Female	04 (50.0%)	04 (50.0%)		

Table-IV. Hyperhomocysteinemia in different age groups

Age	Hyperhomocysteuiemia		
	Yes	No	
21-30	01 (20.0%)	04 (80.0%)	
31-40	26 (43.3%)	34 (56.7%)	

Table-V. Chest pain in different age groups of patients

Sex	Chest	Chest pain	
	Yes	No	
21-30	04 (80%)	01 (20%)	05 (100%)
31-40	57 (95%)	03 (05%)	08 (100%)
Total	61 (93.8%)	04 (6.2%)	65 (100%)

DISCUSSION

Acute MI is a major cause of morbidity and mortality worldwide and elevated level of homocysteine is a popular non-lipid risk factor for the vascular disease. Thousands of articles have been published on the role of homocysteine in cerebrovascular and cardiovascular diseases.

The relative risk of vascular disease for participants in the

top 5th of fasting homocysteine level distribution (>12 μ mol/L) was 22 compared with that bottom four fifth ones⁴.

Total homocysteine includes homocystein and mixed cystinehomocysteine disulfide⁵. Efforts were made to obtain fasting plasma samples to standardize testing conditions and total homocysteine measurement⁶.

Most participants in present study had routine diagnostic tests such as carotid duplex untrasonography and transthoracic echo-cardiography. Previous atrial fibrillation was not exclusionary if an electrocardiogram (ECG performed within the 30 days preceding recruitment showed normal sinus rhythm.

It is clear from our study that frequency of hyperhomocysteinemia is 41.5% which is almost equal to other studies. So far 3 studies have been published regarding hyperhomo-cysteinemia and CAD in Pakistan. While studies by Salahuddin et al and Aamir et al have reported positive association between elevated plasma homocysteine and CAD^{7,8} and the frequency of hyperhomocysteinemia in these studies was about 35-40% which was almost equal to our study. A recent study from the AKUH laboratory showed lack of association between hyperhomocysteinemia and CAD in a Pakistani population of patients with AMI⁹. The difference appears to be due to the design of the studies, size of the population and socio-economic background of the subjects. Mean plasma homocysteine levels in AMI patients and controls in the studies by Salahuddin etal, Aamir et al and Igbal et al were 104.9 ± 1.13 umol/l, 10.57 ± 0.31 umol/l, 18 ± 5.9 umol/l 10.8 ± 4.1 umol/l, 18 ± 8.36 umol/l and 16.4 ± 4.9 umol/L respectively.

Wald et al estimated that reducing total homocysteine by 3 μ mol/L is associated with a 24% reduced risk of stroke (95% CI, 15-33%) and a 16% reduced risk of ischemic heart disease (95% CI, 11-20%). This implies a 13% combined stroke/coronary event reduction for a difference of 2 μ mol/L¹⁰.

The observation that elevated serum Hcy concentration appeared to increase the risk of CVD mortality in smoking men is similar to a recent case control study with In a recent case control study done in 110 subjects and 154 controls in a French population with a low risk of CVD, revealed a significant 29% increased adjusted risk of CVD mortality in the highest plasma Hcy level compared to the lowest one¹².

Our study has some limitations, most important of which is the small sample size, due to which it is not possible to generalize the results, exact significance of the homocysteine level in different co-morbid conditions of the 65 patients cannot be established. Therefore, a large number of patients of different age groups and clinical conditions should be screened for the homocysteine level in order to determine the normal level of plasma homcysteine.

CONCLUSIONS

Plasma Hcy is an important risk factor for the development of the acute myocardial infarction. The mean homocysteine level in patients of AMI was greater than the cut off value of 15 mol/l. furthermore, the difference between male and female ratio greater as compared to other study.

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REFERENCES

- 1. Moschos N, Christoforaki M, Antonatos P. Seasonal distribution of AMI and its relation to acute infections in a mild climate. Int J Cardiol 2004; 93: 39-44.
- Goyal P, Kale SC, Chudhry R, Chauhan S, Shah N. Association of common chronic infections with CID in patients without any conventional risk factors. Ind J Med 2007; 125: 129-36.
- 3. Iqbal MP. Hyperhomocysteinemia and coronary artery disease in Pakistan. J Pak Med Assoc 2006; 56: 282-6.
- 4. Garham IM, Daly LE, Refsum HM. **Plasma** homocysteine as a risk factor for vascular disease. JAMA 1997; 277: 1775-81.
- 5. Refsum H, Smith AD, Ueland PM. Facts and recommendations about total homocysteine

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determinations: an expert opinion. Clin Chem 2004; 50: 3-32.

- Jacques PF, Bosyom AG, Wilson PW. Determinants of plasma total homocysteine concentration in the Framingham Offspring cohort. Am J Clin Nutr 2001; 73: 613-21.
- Aaimir M, Sattar A, Dawood MM, Dilawar M, Ijaz A, Anwar M. Hyperhomocysteinemia as a risk factor for ischemic heart disease. J Coll Phys Surg Pak 2004; 14: 518-21.
- 8. Wald DS, Law M, Morris JK. Homocysteine and cardiovascular disease: evidence on casuality from a meta analysis. Br Med J 2002; 325: 1202-6.

- Iqbal MP, Ishaq M, Kazmi KA, Yousuf FA, Mehbiib AN, Ali SA. Role of vitamins B6, B12 and folic acid on hyperhomocysteinemia in a Pakistani population of patients with AMI. Nutr Metab Cardiovas Dis 2005; 15: 100-8.
- Cook JVV, Taylor J. Lloyd M. Orloff SL. Homocysteine and atrial disease experimental mechanisms. Vasc Pharmacol 2002; 38: 293-300.
- 11. O'Callaghan P, Meleady R, Fitzgerald T, Graham I. **Smoking and plasma homocysteine.** Eur Heart J 2002; 23: 1580-6.
- 12. Blacher J, Benetos A Kirzin JM. Relation of plasma total homocysteine to cardiovascular mortality in a French population. Am J Cardiol 2002; 90: 591-5.

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- Aftab Ahmad, Zafar Hussain Tanvir, Zafar Hussain. ACUTE MYOCARDIAL INFARCTION; SERUM MAGNESIUM AND ELECTROLYTE LEVELS AT PRESENTATION IN EMERGENCY DEPARTMENT (Original) Prof Med Jour 17(2) 246-251 Apr, May, Jun 2010.
- Ishtiaq Ahmed Malik, Khalid Mahmood, M Khalid Raja. ACUTE MYOCARDIAL INFARCTION (Original) Prof Med Jour 12(4) 457-465 Oct, Nov, Dec 2005.
- Liaqat Ali, Abdul Rehman Abid, Imtiaz Ahmed, Nusrat Niaz, Tahira Abdul Rehman, ,Muhammad Azhar. MYOCARDIAL INFARCTION IN DIABETICS; CIRCADIAN PERIODICITY IN THE ONSET OF ACUTE ST SEGMENT ELEVATION (Original) Prof Med Jour 18(2) 269-274 Apr, May, Jun 2011.
- Liaqat Ali, Abdul Rehman Abid, Jahangir Ahmed, Nusrat Niaz, Tahira Abdul Rehman, Muhammad Azhar. MYOCARDIAL INFARCTION; CLINICAL PREDICTORS OF IN HOSPITAL COMPLICATIONS IN PATIENTS PRESENTING WITH ACUTE ST SEGMENT ELEVATION (Original) Prof Med Jour 18(3) 418-425 Jul, Aug, Sep 2011.
- Shahid Hafeez, Asim Javed, Azhar Mahmood Kayani, ACUTE MYOCARDIAL INFARCTION; SIGNIFICANCE OF 1st SET OF CK-MB IN DIAGNOSIS(Original) Prof Med Jour 16(2) 198-201 Apr, May, Jun, 2009.
- Sheikh Nadeem Ahmad, Syed Saud Hasan, Muhammad Yousuf Salat. MYOCARDIAL INFARCTION; STREPTOKINASE STUDY ON ST-SEGMENT RESOLUTION IN PATIENTS AGE LESS THAN 40 YEARS (Original) Prof Med Jour 18(4) 671-677 Oct, Nov, Dec 2011.
- Zafar Iqbal, Tasneem Zafar. ROLE OF LIPOPROTEINS IN MYOCARDIAL INFARCTION (Original) Prof Med Jour 14(1) 82-88 Jan, Feb, Mar, 2007.