Research Journal of Pharmaceutical, Biological and Chemical Sciences

Evaluation of Clinical Utility of Serum Enzymes, Lipid Profile, Homocysteine in Early Stages of Acute Myocardial Infarction.

Ch Sankeerthi[1], A Vaithialingam[2], T Sandhya Rani[3], BS Ravi Kiran[1], T Mohanalakshmi[3],and E Prabhakar Reddy[1].

[1]Department of Biochemistry, Sri Lakshmi Narayana Institute of Medical Sciences, (Affiliated to Bharath University, Chennai), Puducherry, India.
[2]Department of Orthopeadics Sri Lakshmi Narayana Institute of Medical Sciences, (Affiliated to Bharath University, Chennai), Pondicherry, India.
[3]Department of Microbiology, Sri Lakshmi Narayana Institute of Medical Sciences, (Affiliated to Bharath University, Chennai), Pondicherry, India.

ABSTRACT

Coronary artery disease is now a major health problem in India. In past few decade, the battle to reduce the incidence of CAD has led the researchers to look for various clinical markers, which would help early diagnosis of the diseases. The present study was undertaken to assess the levels of cardiac enzymes like Creatine kinase (CK),Creatine kinase isoenzyme MB (CKMB),LDH and Transaminases, lipid profile like Triacylglycerol level, Cholesterol , LDL and Homocysteine in selected Myocardial infarction (MI) patients. Fasting blood samples were taken from 50 MI patients (30 males and 20 females) and compared with age matched controls. Blood samples from MI patients were taken at Zero hours (i.e; at the time of admission),1 hour and after 6 hours. By the end of 6 hrs,CK,CKMB ,LDH,TGL,LDL and Homocysteine increases whereas SGOT,SGPT and cholesterol does not showed significant difference when compared with controls. Thus the present study indicates a strong association between the cardiac enzymes, lipid profile and homocysteine level with the AMI and thus implying possible risk factors for MI.
Keywords: AMI, CAD, Cardiac enzymes, LDL, Homocysteine.

*Corresponding author

INTRODUCTION

Acute myocardial infarction (AMI) is the most important consequences of coronary artery disease.CAD is the most common cause of death. It is responsible for the deaths of approximately one in five and one in six women [1].

MI is now considered as a part of spectrum referred to as Acute Coronary Syndrome.[1] Although traditional risk factors of MI are helpful in diagnosis, specific clinical markers would be valuable in identifying the persons who are at risk [2] .

In the UK, the highest recorded rates of CAD mortality are people born in India, Pakistan, and Bangladesh. South Asians are thought to have a 40-60% higher risk of CHD related mortality compared to other population and this is due to use of ghee in cooking, raised blood glucose, cholesterol, parental consanguinity, paternal history of CVD, low level of income and lack of education [3].

The average incidence of myocardial infarction for those aged between 30 and 69 years is about 600 per 1, 00,000 for men and 200 per 1, 00,000 for women. Incidence increases with age and elderly people also tend to have higher of morbidity and mortality from their infracts [1].

Risk factors may be increasing age, male, family history of premature history of premature CHD, premature menopause. Modifiable risk factors for atherosclerosis include smoking, Diabetes mellitus, metabolic syndrome, hypertension, hyperlipidaemia, obesity and physical inactivity.

Diagnosis of infarction can be achieved by ECG changes and elevation of cardiac enzyme markers. In addition to help diagnosis of AMI, the laboratory parameters are of major importance in monitoring cause of infarction and in estimating its size nowadays highly playing important role in diagnosis of AMI [4].

Cytosol proteins such as CK are washed out in different percentage in dependence on the blood flow in the earlier phase of infarction and therefore appears earlier in the reperfused infarct. Although with certain limitations the differences in the release kinetics of CK can be used to evaluate the outcome of thrombolytic treatment, the perfusion dependent washout of cytosol proteins, once the reperfusion has occurred [5]. In addition to CK other biochemical parameters like LDH, SGOT, SGPT also shows abnormal levels during AMI.

The pathogensis of AMI is multi factorial however, several studies have implicated impaired lipid metabolism as one of the crucial factors in the development of the disease [6]. So much attention has been focused on serum lipid profile because of their strong association of CAD.

The above literature clearly indicates important role of lipid metabolism in AMI. However, biomarker value of various components of lipid profiles is not clear due to

conflicting findings in various studies. We therefore compared the lipid profile of AMI with respect to normal subjects [6].

There are some cases with MI those who don't have any of the traditional risk factors .So, attention has been focused on other predisposing factors which may contribute to MI. Researchers have taken effort to find the possible association between Plasma Homocysteine levels in AMI [7]. Several studies conducted in different parts of the world was reported the elevated levels of Plasma Homocysteine are associated with CAD, independent of other risk factors.

We therefore compared the cardiac enzymes, lipid profiles and homocysteine levels in AMI with respect to normal subjects. Therefore this has been taken due to the incidence of mortality in MI is high.

MATERIALS AND METHODS

50 patients out of which 30 ales and 20 females admitted to the intensive care unit of Sri Lakshmi Narayana Medical College, Pondicherry with documented AMI showing characteristic ECG signs and rise in troponin I concentrations were selected and compared with 50 age matched controls who were volunteers in our hospital.

The patients who were under recent surgery, infections, chronic inflammatory diseases, significant hepatic or renal dysfunction and malignanacy were excluded from the study.

Sample collection

Fasting blood was collected from the patients within 6 hrs from the onset of the infarction. Plasma was separated from collected blood sample using EDTA coated tubes. Serum was separated by centrifugation at 2000 rpm and is used for the estimation of cardiac enzymes like CK,CKMB,LDH,SGOT,SGPT and lipid profile such as total cholesterol, TG's, and LDL. The separated plasma was used for analyzing the plasma homocysteine using ELISA.

The values of cardiac enzymes, lipid profile and homocysteine levels in patients of AMI were compared with the normal subjects.

RESULTS

Myocardial infarction can occur at any age irrespective of sex, but generally it is a disease of middle and old age. The result of the present study is given as below.

Table 1   Serum enzymes

Parameters	Males (30)	Female (20)	First hour	After 6 Hrs	Controls	P value
CK	212.3±1.82	286±2.08	192±1.34	298±2.10	188±1.23	< 0.0001
CKMB	30.32±1.79	32.36±1.89	24±1.34	38.23±1.92	18±1.29	<0.001
LDH	442±3.02	458±3.82	-	-	286±3.02	<0.001
SGOT	28.6±2.98	30.2±3.02	28.38±2.93	32.2±3.23	28.20±2.92	<0.001
SGPT	29.20±2.88	29.18±2.88	29.18±2.87	29.30±2.92	29.12±2.86	<0.026

Table 2   Lipid profile and Homocysteine

Parameters	Males (30)	Female (20)	First hour	After 6 Hrs	Controls	P value
Cholesterol	177.07±8.49	183±8.75	-	-	188.5±8.38	<0.001
TG	131.23±12.54	139.26±12.81	-	-	117.7±12.34	<0.001
LDL	143.4±.6.84	153.1±6.83	-	-	115.03±2.38	<0.001
Homocysteine	16.56±1.12	18.91±1.83	-	-	12.40±0.99	<0.001

DISCUSSION

The diagnosis of acute myocardial infarction (AMI) as traditionally been based on the characterstic clinical history, ECG abnormalities and increased serum concentrations of cardiac marker enzymes. As the differentitial diagnostic value of chest pain is limited and the ECG changes have various degrees of sensitivity and specificity, the measurements of serum enzymes as a reflection of damage to myocardial muscle cells still play an important role in the diagnosis of AMI. Measurements of CK,LDH,SGPT,SGOT are well established methods.

The Creatinine kinase value in males is 212.3±1.82 and in females 286±2.03.the level increases in AMI patients at first hour and after six hours when compared with the controls 188±1.29 where the P value <0.0001.

In our present study, the LDH values in males is 442±3.02 whereas females 458±3.82 which shows a significant raise when compared with the controls 286±3.02. Further in our study the pattern of changes in the concentration of SGOT and SGPT showed no variation from that of CK and CKMB. Both SGOT and SGPT were normal at zero hours (at the time of admission) there was no raise at the end of 1st hr and after 6 hrs. These observations of our study comparable to the studies reported by other authors.

In case of lipid profile, our results showed significant increase in TG and LDL levels in AMI patients but the cholesterol level shows no significant raise as compared with the controls.

The mechanism of increase in TG's after acute MI may be due to elevated flux of fattyacids and impaired removal of VLDL from the plasma.[8] Another possible mechanism for elevated TG levels may be the effect of β blockers but this seems to be invalid for increased TG levels on day 3 and pre-discharge (day 7) as β blockers take about two weeks to show their effect on serum lipids [8].

LDL cholesterol recorded high significant changes throughout the study period in our subjects. However a significant decrease or no significant change in LDL cholesterol following MI has been reported by others [10].

We found no significant changes in serum total cholesterol levels either during the AMI or after 3 months. Our study provides information which is direct contrast to that by others who found either a decrease or increase during the acute phase of MI [8,11].

There is an elevation of Plasma Homocysteine in AMI patients when compared with control group (table 2). Statistical significant difference were observed in the mean of Plasma Homocysteine concentrations between the AMI patients (male 16.56±1.12 mM/L, females 18.91±1.83) and in controls (12.40± 0.99mM/L) [6].

Recent report on Homocysteine suggests that it is an independent predictor of vascular disease including stroke and coronary artery disease. Mutations in the methyl tetrahydrofolate reductase gene are one of the most frequent causes of moderatively elevated plasma Homocysteine [6]. Hyperhomocysteinemia may also occur due to nutrition deficiency that leads to low blood concentration of folate, Vit B_{12} or vitamin $B_6.$

Homocysteine may also induce atherosclerosis by effecting endothelial derived relaxin factor Nitric oxide (NO). It also enhances the lipid peroxidation which may decrease the expression of endothelial NO synthase and directly degrade NO. The finding of this study underscores the importance of determining the levels of Plasma Homocysteine in individuals who are at risk of developing AMI [3].

CONCLUSION

In Conclusion, our data provides clear evidence that serum enzymes, lipid profiles and Homocysteine levels are markedely useful in determining the patients of Acute Myocardial Infarction.

REFERENCES

[1]   Acute myocardial infarction.www.doctor AMI/Doctor/Patient.co.uk
[2]   T Angeliene, et al. Indian J Clin Biochem 2003;18(1):103-106.
[3]   www.ncbi.nl.nih.gov/--/PMC1768096
[4]   Hitesh shah and N Haridas. Indian J Clin Biochem 2003;18(2):93-101.
[5]   Hitesh Shah and N Haridas. Indian J Clin Biochem 2007;22(1):33-36.
[6]   Haseeb A Khan, Abdullah S Alhomida and Samia H Sobki. Biomarker Insights 2013;8: 1–7
[7]   T Angeliene, et al. Indian J Clin Biochem 2005;20(1):18-20
[8]   PK Nigam, VS Narain and M Hasan. Indian J Clin Biochem 2004;19(1):67-70.
[9]   Myocardial infarction - wikipwedia
[10]   Prasad K. Int J Angiol 1999;8:76-86.
[11]   Ueland PM, Refsum H, StablerSP.,Malinow MR, Andersson A, Allen RH. Clin Chem 1993;39:1764-1779
[12]   AM Huggon, J Chambers, N Nayeem, P Tutt, MCrook, S Swaminathan. 2014,15-19