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Biochemical Markers and Myocardial Infarction.

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ABSTRACT

Myocardial infarction (MI) continues to be a major cause of mortality and also death at early ages all over the world. This study is undertaken to evaluate the role of biocardiac markers in MI and to identify some reliable markers which would help the physician in the early stages diagnosis enabling him to intervene effectively to reduce subsequent mortality rates. Lipid profile, lipid ratios, CK-MB and Troponin I are measured in patients with chest pain. Troponin I is seen as an ideal cardio sensitive and specific marker for the early diagnosis of Myocardial infarction. A combination of CK-MB and Troponin I is further confirmation of MI. Measurement of lipid profile and lipid ratios from an early age and correction of dislipidemia would help to reduce the incidence of MI and to prevent early age deaths .Further, through curtailing health care expenses primary health care could be extended to more of the needy and at affordable cost.

KEYWORDS Cardiospecific markers, Troponin, CK-MB, Lipid profile and Lipid ratios in MI.

INTRODUCTION

Cardiovascular diseases and stroke are the major contributors to mortality and morbidity worldwide resulting in large number of premature deaths. It is estimated that about two thirds of the globally estimated 14.3 million annual cardiovascular diseases occur in the developing countries. By the year 2015 cardiovascular disease could be the major cause of mortality in India. (1).

Serum cardiac biomarker parameters play a major role in diagnosing acute myocardial infarction. They also monitor the course and extend of infarction (2, 3). Number of genetic and environmental factors is alleged to be the causes of coronary heart diseases (4). Dislipidemia, hypertension, diabetes and obesity are the genetic causative components while life style behavior such as smoking, dietary habits and lack of physical exercise are modifiable factors (5). Low plasma glucose, high CK-MB, elevated and altered lipid profile are the biocardiac markers associated with MI. Among these CK-MB and Troponin I are sensitive and specific cardiac markers and assist the physicians in the early diagnosis of MI. Contradicting reports exist regarding kidney function tests in MI patients. In the present study the role of biomarkers – lipid profile, lipid ratios, kidney function tests, CK-MB and Troponin I are estimated in MI patients and compared with controls.

MATERIALS AND METHODS

Male and female MI patients of IPD of SK Hospital form the core of the study. Patients presenting with acute chest pain suggestive of acute MI and who are so diagnosed by expert cardiologists on the basis of patient history, symptoms and abnormal ECG are selected for the study.

EXCLUSION:

Patients with sepsis pericarditis, pulmonary embolism and renal failure are excluded from this study.

METHODS:

Total cholesterol by CHOD and POD method.

HDL Cholesterol estimated by direct measurement using two reagent formats.

LDL –Cholesterol measured directly in a tow reagent format.

Triglycerides by a combination of enzymes-Lipoprotein lipase, glycerokinase and preoxidase.

VLDL-Cholesterol is calculated by Fredrikson's equation.

Blood Urea Nitrogen (BUN)-by glutamate dehydrogenase method.

Creatinine by Jaffe's method.

CKMB is estimated by CMIA technology with flexible assay protocols.

Troponin I measured by flexible chemiflex assay.

Troponin I and CK-MB measured using ARCHITECT i1000SR of ABBOTT and all others using automated chemistry analyzer Dimension X pand plus of SIEMENS.

RESULTS

The levels of Total cholesterol (TC), triglycerides (TG) and very low density lipoprotein cholesterol (VLDL) are elevated while high density lipids cholesterol (HDL) levels are decreased in MI patients compared to controls. The differences are statistically highly significant. However there is no significant difference in case of LDL cholesterol between the two groups. However the TC/HDL cholesterol, VLDL-C/HDL-C cholesterol and TG/HDL cholesterol ratios are different between MI patients and controls and these differences are statistically significant.(Table No:1)

Table -2 shows the mean age and lipid profile in females with and without MI. Statistical significance is noticed in case of TC and TG, but not in case of LDL cholesterol, HDL cholesterol and VLDL cholesterol. But all the three ratios TC/HDL cholesterol, LDL/HDL cholesterol and TG/HDL cholesterol are elevated significantly .Hence the lipid ratios are better indicators of risk for MI than lipid levels as such as seen both in males and females.

The cardiac specific and sensitive markers, TroponinI and CK-MB are measured in male controls and male patients and presented in Table 3 .They are elevated significantly in MI patients as compared to controls.

Urea levels are generally higher in MI patients as compared to controls and this rise is not statistically significant in male patients. But, this rise in urea levels is statistically significant in female patients. Creatinine levels are found to be higher both in male and female patients and this increase is statistically significant. Hence it is justified to conclude that kidney function is impaired in MI patients (Table Nos: 3, 4)

DISCUSSION

Acute myocardial infarction is the major cause of premature disability in developing societies (7). Serum cardiac biomarkers analysis are cumbersome processes in the detection of MI.

There is a rise in the level of Total cholesterol, Triglycerides, LDL cholesterol and VLDL cholesterol and a fall in HDL cholesterol in MI patients as compared to controls. Increased LDL cholesterol in MI patients as compared to controls has been recognized as an independent risk factor by NCEP (8).The elevated levels of LDL cholesterol can lead to increased levels of oxidized LDL cholesterol which enhances the atherogenisity in the coronary vessels. Oxidized LDL cholesterol possesses chemostatic and cytotoxic effects which result in the formation of foam cells that play a major role in the phenomenon of atherosclerosis.

Elevated synthesis of apoB by liver increases production of VLDL and LDL which leads to increased turnover of TG from VLDL to LDL .The triglycerides rich LDL undergoes hydrolysis producing dense LDL which are more atherogenic because of greater arterial wall retention and increased susceptibility to oxidized VLDL. Further it forms an effective substrate for free radical generation and the resultant products could accelerate the progression of the cardiac diseases.

In MI if the ischemia persists for a long time it causes severe damage to the heart muscles. Estimation of serum CK-MB is helpful in diagnosis and progression of therapy.

There is significant increase in the levels of CK-MB and Troponin I in MI patient as compared to controls. Of the biocardiac markers TroponinI is the cardiac specific and sensitive biomarker which can help to diagnose MI at its early stages.

In myocardial infarction if the ischemia persists for long it cause severe damage to the heart tissue. There is also a significant increase in the CK-MB levels .The values of CKMB are elevated 4 to 6 hours after the onset of MI .In the present study CK-MB is elevated 4 to 5 times as compared to controls. This is in agreement with the reports of Robert.R.Ester et al(9) and John.J and Fenton et al(10).

CONCLUSIONS

The estimation of serum Troponin I is valuable in the diagnosis of all cases with signs and symptoms of chest pain.

Combined use of cardiac Troponin I along with ECG and clinical history of chest pain will help in the early and accurate diagnosis and initiation of treatment .Under circumstances where Troponin I assay is not possible, then cardio specific and sensitive CK-MB can be assayed.

Myocardial infarction is the leading health problem all over the world .Identification of major risk factors and awareness through population based strategies for prevention alone can effectively reduce the mortality and morbidity rates.

MI imposes a heavy burden over community in terms of social, economic and physiological terms. Being a major health care problem, it has become a major financial burden whereever primary health care offered is free of cost to the public. Hence the ideal policy will be to evaluate the biomarkers as a routine procedure to prevent /reduce the incidence of MI .The CK-MB and Troponin I being specific and sensitive cardiac markers will be elevated after onset of MI .The other biomarkers such as lipid profile and lipid ratios are also indicative of high risk for MI . Public awareness of these factors is of paramount importance in the control and progression of MI. Early diagnosis and effective intervention go a long way in reducing the risk to life, minimizing the complications in delayed diagnosis and providing timely medical care at affordable cost even to the common masses. Owing to the ever increasing burden caused by the MI over the community, it is imperative task for the health care professionals as well as governments to diagnose and treat it well in advance and curb its increase in the community for which identification and measurement of risk factors and biochemical markers for cardiac diseases such as lipid profile is a must .Early measurement of biomarkers will help the physician to minimize the complications due to progression of the diseases.

TABLE NO: 1
AGE AND LIPID PROFILE IN MALE CONTROLS AND MI PATIENTS

		NUMBERS	MEAN±SD	P value
Age	Case	50	65.42 ±10.88	0.373
	Control	50	63.72±7.88	
TC	Case	50	182.24±31.82	<0.001
	Control	50	139.16±19.2	
TG	Case	50	164.64±33.78	<0.001
	Control	50	112.02±18.19	
LDL-C	Case	50	95.42±15.65	0.823
	Control	50	94.8±11.7	
HDL-C	Case	50	31.1±5.17	<0.001
	Control	50	40±4.69	
VLDL-C	Case	49	33.51±8.6	<0.001
	Control	50	21.26±3.94	
TC/HDL-C	Case	50	5.9±0.78	<0.001
	Control	50	3.49±0.34	
LDL/HDL	Case	50	3.09±0.35	<0.001
	Control	50	2.39±0.31	
TG/HDL	Case	50	5.3±0.61	<0.001
	Control	50	2.82±0.44	

TABLE NO: 2
LIPID PROFILE AND LIPID RATIOS IN FEMALE MI PATIENTS AND CONTROLS

		NUMBERS	Mean±SD	P value
TC	Case	30	203.0±43.3	<0.001
	Control	30	143.5±16.0	
TG	Case	30	163.8±31.1	<0.001
	Control	30	114.4±11.2	
LDL-C	Case	30	128.0±9.1	0.025
	Control	30	89.6±9.3	
HDL-C	Case	30	43.1±60.8	0.853
	Control	30	41.0±4.9	
VLDL-C	Case	30	42.9±62.9	0.104
	Control	30	23.8±5.2	

TC/ HDL-C	Case	30	6.3±1.9	<0.001
	Control	30	3.5±0.3	
LDL/HDL	Case	30	3.9±2.4	<0.001
	Control	30	2.2±0.2	
TG/HDL	Case	30	5.1±1.7	<0.001
	Control	30	2.8±0.3	

TABLE NO: 3
UREA, CREATININE, TROPONIN I AND CKMB LEVELS IN MALE MI PATIENTS AND CONTROLS.

		NUMBERS	Mean±SD	P value
Urea	Case	50	39.32±7.58	0.623
	Control	50	24.56±6.45	
Creatinine	Case	50	1.57±0.44	<0.001
	Control	50	1.11±0.23	
Troponin I	Case	50	20.52±10.2	<0.001
	Control	50	0.07±0.08	
CKMB	Case	50	10.85±9.6	<0.001
	Control	50	1.69±0.52	

TABLE NO:4
UREA, CREATININE, TROPONIN I AND CKMB LEVELS IN FEMALE MI PATIENTS AND CONTROLS.

		NUMBERS	Mean±SD	P value
Urea	Case	30	41.3±5.0	<0.001
	Control	30	23.1±6.1	
Creatinine	Case	30	1.6±0.7	<0.001
	Control	30	1.0±0.2	
Troponin I	Case	30	12.2±16.5	<0.001
	Control	30	0.1±0.1	
CK-MB	Case	30	17.8±12.6	<0.001
	Control	30	1.6±0.4	

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