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Correlation between CK-MB, TSH, LDL, HDL, Troponin T, Troponin I and myocardial infarction

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Abstract---Introduction: The assessment of patients with acute chest pain of possible cardiac cause continues to be a challenge and positive diagnosis has psychological, social and legal implications. The presence of ST segment elevation in the ECG is highly specific (but only about 50% sensitive) for acute myocardial infarction (MI). However, many patients presenting to coronary care units has chest pain without ST elevation in the ECG. The diagnostic possibilities in these cases include: acute coronary syndrome in evolution, or 'non-ischaemic' chest pain. The World Health Organization defines for diagnosing AMI, the presence of two of the three enlisted features namely, symptoms of myocardial ischemia, elevated levels of cardiac marker (protein or enzyme) concentrations in the blood, and a typical electrocardiographic pattern involving the development of Q waves or persistent T wave changes. Methods: In this Prospective study, the data of the registry maintained in the Department of biochemistry, Tertiary care Teaching Hospital were analysed. The WHO case definition was used to assign a diagnosis in 430 patients presenting to the emergency department with symptoms of cardiac ischemia. The inclusion criteria were the subjects (n=430) who were admitted to the intensive care unit of the hospital complaining severe chest pain and who were requested by the medical staff to get both troponin and lipid profiles done. Result: In Table 1 showed mean value of Age and BMI was significantly increase in patients with myocardial infarction compared with control groups. The results showed that significantly increased in Cholesterol, TG, LDL and VLDL also significantly decreased in HDL in patient with myocardial infarction when compared to control groups Table 2. Conclusion: Patients with chest

pain and positive troponin test (with confirmed cardiac event) were found to have significantly elevated levels of total cholesterol, triacyl glycerol levels, low density lipoprotein level and significantly reduced high density lipoproteins cholesterol levels when compared to the patients who experienced only chest pain (negative troponin) and healthy controls.

Keywords---high-sensitivity cardiac troponin T, troponin I, myocardial infarction, CKMB, TSH, LDH.

Introduction

The assessment of patients with acute chest pain of possible cardiac cause continues to be a challenge and positive diagnosis has psychological, social and legal implications. ^[1] The presence of ST segment elevation in the ECG is highly specific (but only about 50% sensitive) for acute myocardial infarction (MI). However, many patients presenting to coronary care units has chest pain without ST elevation in the ECG. The diagnostic possibilities in these cases include: acute coronary syndrome in evolution, or 'non-ischaemic' chest pain. ^[2]

The World Health Organization defines for diagnosing AMI, the presence of two of the three enlisted features namely, symptoms of myocardial ischemia, elevated levels of cardiac marker (protein or enzyme) concentrations in the blood, and a typical electrocardiographic pattern involving the development of Q waves or persistent T wave changes. ^[3] Traditionally the cardiac enzymes used in the assessments for the detection of MI includes the triad of lactate dehydrogenase, aspartate transaminase (serum glutamate oxaloacetate transaminase) and creatinine kinase-MB (CKMB) which is of heart origin. However, the use of biochemical 'gold-standard' CK-MB levels has limited prognostic power compared to the serum troponins (both I and T) which are considered to be more specific and sensitive over CK-MB in the setting of acute coronary syndromes and have been validated for post operative risk stratification for noncardiac surgical procedures. ^[5]

Highly sensitive and specific immunoassays for myocardial proteins, such as troponins T and/or I which are components of the thin filaments of the sarcomere are used in the identification of subjects with small areas of myocardial necrosis. ^[6] The magnitude of troponin elevations has correlated with the risk of death and composite risk of death or non-fatal MI, irrespective of whether the patients had ST elevation or non-ST elevation acute coronary syndromes by observations of the recent studies. ^[7]

Troponin I testing had better sensitivity, specificity and prognostic value than troponin T testing. A positive troponin I result was a strong predictor of cardiac events (death from cardiac causes or MI) in the next 30 d. The predictive value of a negative troponin I result was also high, with a total 30 d event rate of 0.3%, regardless of the admission ECG. The new diagnostic criteria include a characteristic rise and fall in blood concentrations of cardiac troponins and/or CK-MB in the context of spontaneous ischemic symptoms or coronary

intervention. [8] Cardiac troponin I and T are highly sensitive and highly specific and may be elevated when CKMB concentrations are not even mildly elevated. In addition, they may predict recurrent cardiac events in patients with acute coronary syndromes. However, use of troponin testing has been limited by availability of laboratory-based diagnostic techniques and by relatively long processing times. [9]

Methods

In this Prospective study, the data of the registry maintained in the Department of biochemistry of the Tertiary care Teaching Hospital were analysed. The WHO case definition was used to assign a diagnosis in 430 patients presenting to the emergency department with symptoms of cardiac ischemia. The inclusion criteria were the subjects (n=430) who were admitted to the intensive care unit of the hospital complaining severe chest pain and who were requested by the medical staff to get both troponin and lipid profiles done.

In addition to that, reports of 165 healthy subjects who had got their lipid profiles checked using the medicare facility were assessed as controls. The troponin was detected qualitatively when a specimen contains troponin I (ctni) above the 99th percentile (TnI>0.5ng/ml) method. The total cholesterol (TC), high density lipoproteins cholesterol (HDL), very low-density lipoproteins (VLDL), and triacyl glycerol levels (TG) were analysed, using the kits provided by HUMAN Diagnostics and the low-density lipoprotein level (LDL) was calculated using Fried Ewald formula.

All the estimations were done using HUMAN 300 semiauto analyser and data was analysed using Epi Info windows version. Significance of the difference of parameters among different groups was analysed using Z-test. The reports of the subjects with any of the missing data were excluded. The selection of the reports was done without the prior knowledge of both the subjects and the staff of the intensive care unit. So that healthcare workers and the study subjects were not influenced anyway during the study. Therefore, no written consent was obtained from any of the subjects.

Result

In table 1 showed mean value of Age and BMI was significantly increase in patients with myocardial infarction compared with control groups.

Table (1): Mean value of Age and BMI in myocardial infarction and control groups

	Control Mean \pm SD	Patients Mean \pm SD	p-value
Age	38.17 \pm 6.22	58.35 \pm 7.64	0.001
BMI (kg/m ²)	22.17 \pm 3.41	31 \pm 5.42	0.002
*Significant using SPSS for two independent means at significance * (P \leq 0.05), ** (P \leq 0.01)			

Table (2): Mean value of Lipid profile in myocardial infarction and control groups

Parameters	Control Mean \pm SD	Patients Mean \pm SD	p-value
Ch (mg/dl)	117.82 \pm 18.56	251.82 \pm 37.18	0.001
TG (mg/dl)	93.64 \pm 4.65	161.82 \pm 101.54	0.032
HDL (mg/dl)	54.37 \pm 3.94	41.37 \pm 3.91	0.001
LDL (mg/dl)	78.64 \pm 16.09	167.19 \pm 42.41	0.000
VLDL (mg/dl)	18.37 \pm 0.09	38.54 \pm 22.72	0.002
*Significant using SPSS for two independent means at significance * (P \leq 0.05), ** (P \leq 0.01)			

Table (3): Mean value of T3, T4 and TSH in myocardial infarction and control groups

Parameters	Control Mean \pm SD	Patients Mean \pm SD	p-value
T ₃ (ng/ml)	1.06 \pm 0.09	2.12 \pm 0.09	0.009
T ₄ (mg/dl)	7.08 \pm 0.57	9.37 \pm 0.55	0.001
TSH (mIU/l)	1.57 \pm 0.64	1.23 \pm 0.98	0.421
CK-MB (ng/mL)	1.24	3.56	0.001
Troponin T (ng/L)	3.2	6.3	0.001
Troponin I (ng/L)	1.6	3.9	0.001
*Significant using SPSS for two independent means at significance * (P \leq 0.05) and ** (P \leq 0.01)			

The study showed levels of T₃ and T₄ was significantly increased while TSH decreased in patient with myocardial infarction when compared to control groups as shown in Table (3).

Table (4): Baseline Pearson relation coefficients of T₄ levels with various lipid profile (Ch, HDL, VLDL) in myocardial infarction and control groups

Correlation	Ch	HDL	VLDL
T ₄	0.994 0.001	-0.886 0.002	0.887 0.002
* Correlation is significance * (P \leq 0.05), ** (P \leq 0.01)			

Levels of T₄ with (Ch, VLDL) showed positive significant correlation coefficient also there is negative correlation coefficient between T₄ with HDL in patients with myocardial infarction and control groups as shown in Table (4) and Figure (1,2,3).

Discussion

Hyperthyroidism is the clinical syndrome caused by increase of circulating free Thyroxine T₄, free Triiodothyronine T₃, or both. It is a common disorder that affects approximately 2% of women and 0.2% of men. Also, the diagnosis of hyperthyroidism is confirmed by blood tests that show decreased of thyroid stimulating hormone (TSH). [10]

The results showed that significantly increased in Cholesterol, TG, LDL and VLDL also significantly decreased in HDL in patient with myocardial infarction when compared to control groups Table (2). High serum cholesterol level considered as a risk factor for cardiovascular disease. Also, Triglyceride another strong risk factor but it found that triglyceride levels stratifying led to more accurate detection of increased risk of coronary disease. In addition, elevated LDL is associated with 3-fold increase in the risk of myocardial infarction. Increased VLDL in Myocardial infarction patients and the role of low HDL in the CHD development has been widely accepted. ^[11]

The cardiovascular manifestations of hyperthyroidism have been recognized for more than two centuries and are a cornerstone for clinical diagnosis. The peroxidation of lipids is basically damaging because the formation of lipid peroxidation products leads to spread of free radicals' reactions. ^[12-14] Where hydroxyl radical can initiate lipid peroxidation, which is a free radical chain reaction leading to damage of membrane structure and function. Free radical mediated oxidative stress (OS) implicated in the pathogenesis of thyroid disorders. ^[15] In addition, Free radicals have the potential to damage the organism, their generation is inevitable for some metabolic process. Variations in the levels of thyroid hormones can be one of the main physiological modulators of in vivo cellular oxidative stress and due to their known effects on mitochondrial respiration. That thyroid hormones are involve in combating the toxicity of oxidative stress in humans. ^[16]

Conclusion

Patients with chest pain and positive troponin test (with confirmed cardiac event) were found to have significantly elevated levels of total cholesterol, triacyl glycerol levels, low density lipoprotein level and significantly reduced high density lipoproteins cholesterol levels when compared to the patients who experienced only chest pain (negative troponin) and healthy controls.

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