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Acute Myocardial Infarction associated with Lipid Profile among Patients at Nasiriyah Heart Center

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Abstract:

The present study is aimed to investigate the relationship between lipids profile and myocardial infarction. Blood samples (130) were obtained from adult human and divided into three groups. The first group (N:44) divided into male (22) and females (22) as a control group. The second group was male patients (N:46), and the third group was female patients (N: 40) as a reviewer of the Nasiriyah Heart Center.

The current results showed a significant increase (P≤0.05) in the concentrations of troponin (Tn) and creatine kinase (CK) in the second group (patients) compared to the control while, the current results showed a significant increase in Creatine Kinase Myocardial Band (CK-MB) in (male patients) compared with (female patients).

The results showed a significant increase in level of Triglyceride (T.G), Low Density Lipoproteins (LDL) and Very Low Density Lipoproteins (VLDL) (P≤0.05) in (male) compared with the control group, while the results showed a non-significant of Total Cholesterol (TC) concentration in (control) and (male patients). Also, there was a significant decrease of High Density Lipoproteins (HDL) level (P≤0.05) in the males compared with the control group. The results showed a significant decrease in TC and LDL level (P≤0.05) in (male patients) compared with the second group (female patients). Also, the results showed that there was non-significant in T.G, HDL and VLDL in males and females patients.

Keywords\ Myocardial Infarction, Troponin, creatine kinase, lipid profile.

Introduction:

Myocardial Infarction (MI) is a term which is used for defining the necrosis in the heart muscle due to the lack of the oxygen of myocardium which cannot be supplied by the coronaries. The pathogenesis of acute myocardial infarction (AMI) is multifactorial; however, several studies have implicated impaired lipid metabolism as one of the crucial factors in the development of this disease [(1)].

The risk of AMI was associated with an increase in LDL and a decrease in HDL, in both Asians and non-Asians.3 Lower concentrations of serum HDL and higher serum TG were found to be independent risk factors [(2)]. Myocardial infarction characterized by chest pains or discomfort which may travel into the shoulder, arm, back, neck or jaw [(3, 4)]. Approximately, 90% of myocardial infarction consequences from an

acute thrombus that barricades an atherosclerotic coronary artery [5]. Some disease factors contribute to the risk of myocardial infarction such as diabetes mellitus (type 1 or 2), high blood pressure, dyslipidemia/hypercholesterolemia, and predominantly high amount of low-density lipoprotein, low amount of high density lipoprotein, high triglycerides, and obesity [4, 6].

There are some cardiac markers that can be used in the diagnosis of myocardial infarction such as Creatine Kinase Myocardial Band (CK-MB) is the enzyme used as a ultimate serum marker for the diagnosis or barring of acute myocardial infarction [4], and second marker are troponins I and T that have the preferred specificity, they have changed the cytosolic enzymes in the role of diagnosing myocardial ischemia and infarction. The use of the troponins provided new knowledge that led to revision and redefinition of ischemic myocardial injury as well as the introduction of biochemical for approximation of the likelihood of future ischemic myocardial actions [7].

The increase of cardiovascular risk derivative from raised cholesterol is advanced and continuous for concentrations above 200mg/dl. Low-density lipoprotein cholesterol (LDL-c) plays an atherogenic role whereas c-HDL acts as a protecting factor [8].

European guidelines published in 2011 reflect that patients with a record of myocardial infarction (MI) are very high cardiovascular risk patients and establish a LDL-c target of less than 70mg/dl or a decrease of basal levels of 50%. Furthermore, they recommend lifestyle changes involving increased physical activity, weight control and quit smoking. Moreover, treatment must be started with the most suitable status according to its effectiveness and basal LDL-c and if not contraindicated, at high doses. Furthermore, patients with triglyceridemia >200mg/dl need non-HDL cholesterol hold of < 100 mg/dl [9, 10].

The current study clearly indicates an important role of lipids metabolism in AMI patients.

MATERIALS AND METHODS:

Subjects

The present study consisted of 130 individuals, 44 of whom were volunteer controls with no clinical evidence of any type of diseases. The remaining 86 individuals were patients with suspected MI admitted to the coronary care unit (CCU) in order to diagnosis and receive treatment, the diagnosis of MI was confirmed by clinical and laboratory findings of chest pain, electrocardiogram changes, and elevations of biomarkers indicating cardiac injury.

This work was carried out during the period from December 2018 to April 2019 in Nasiriyah Heart Center, Thi-Qar, Iraq.

Samples

Three ml of blood was collected via a traumatic antecubital venipuncture into vacutainer tube in the morning after overnight fasting from each subject. The serum was recovered by centrifugation at 3000 rpm for 15 minutes. The samples were used for estimation of serum Tn-I, CK-MB, lipid and lipoprotein profiles. Methods

Methods

A-Determination of serum cardiac Tn-I:

The finecare ^{TM} cTn I rapid quantitative test (cTn I Rapid Quantitative test -Guangzhou wondfo biotech Co, Guangzhou, China) is based on fluorescence immunoassay technology [11]. When sample is added into the sample well of the test cartridge, the fluorescence labeled detector anti cTn I antibodies on the sample pad bind to cTn I antigens in blood specimen and they form immune complexes.

As the complexes migrate on the nitrocellulose matrix of test strip by capillary action, the complexes of detector antibodies and cTn I are captured to anti cTn I antibodies that have been immobilized on test strip.

Thus the more cTn I antigens in blood specimen, the more complexes accumulated on test strip. Signal intensity of fluorescence of detector antibodies reflects the amount of captured cTn I.

B-Determination of serum CK-MB:

Humazym M- Test (Humazym CK-MB-Human Gesellschaft für Biochemica and Diagnostica mbH, Wiesbaden, Germany) is based on an enzymatic CK determination accompanied by an immunoinhibition method [12, 13]. An antibody is incorporated into the reagent which will bind specifically to the M-subunit, inhibiting the enzymatic activity of that subunit. Thus only the remaining activity of the B -subunit is measured. Due to negligible concentrations of CK-BB in the circulation, the remaining activity, multiplied by the factor 2, represents the activity of the CK-MB isoenzyme.

C- Determination of serum lipid and lipoprotein profiles:

Serum lipid and lipoprotein profiles were determined spectrophotometrically by using Human Gesellschaft für Biochemica und Diagnostica mbH kits (Germany). Fasting serum total cholesterol (TC), and T.G were measured according to the method of Trinder [14]. Fasting serum HDL-C was determined by a method described by Gordon [15] while serum fasting LDL-C and VLDL-C were calculated according to Friedewald equation [16]

Statistical Analysis:

Statistical analysis was performed using (SPSS version 23) the T-Test taking ($P \leq 0.05$) as the lowest limit of significance. P value ≤ 0.05 was regarded as statistically significant.

Result:

Table (1) indicated a significant increase ($P \leq 0.05$) of troponin and creatine kinase concentrations in the second group (patients) compared with the first group (control).

Table (1) the relationship between troponin and creatine kinase concentrations in the patients group compared with control group

Parameters / groups	Troponin I (ng/ml)	CK-MB (U/l)
First group (Control)	0.06 ± 0.08^{b}	6.36 ± 2.47^{b}
Second group (Patients)	4.68 ± 0.39^{a}	20.97 ± 1.16^{a}
P- value	0.000	0.000

- Values are means ± S.D.
- Different letters refer to a significant difference ($p \leq 0.05$).
- Same letters refer to non a significant difference ($p \leq 0.05$).

Table (2) there is a non-significant (P≤0.05) of troponin concentrations in the first and second group for patients, while there was a significant increase in the first group compared with the second group.

Table (2) showed relationship between troponin and creatine kinase concentrations in the patients group

Parameters / groups	Troponin I (ng/ml)	CK-MB (U/l)
First group (Male patients)	5.00 ± 0.16[a]	25.79 ± 9.89[a]
Second group (Female patients)	4.32 ± 0.44[a]	16.78 ± 10.58[b]
P- value	0.62	0.00

- Values are means ± S.D.
- Different letters refer to a significant difference (p≤ 0.05).
- Same letters refer to non a significant difference (p≤ 0.05).

The results showed a non-significant (P≤0.05) of TC concentration in the first (control) and second (patients) groups. There was a significant increase (P≤0.05) in level of T.G, while there was a significant decrease (P≤0.05) of HDL level in the second group (patients) compared with the control group. Also, there was a significant increase in LDL and VLD in the second group (patients) compared with the control group table (3).

Table (3) showed relationship between lipid profiles in the patients group compared with control group

Parameters / groups	TC (mmol/L)	T.G (mmol/L)	HDL (mmol/L)	LDL (mmol/L)	VLDL (mmol/L)
First group (Control)	3.91 ± 0.67[a]	1.37 ± 0.26[b]	1.40 ± 0.20[a]	1.87 ± 0.68[b]	0.62 ± 0.11[b]
Second group (Patients)	4.09 ± 1.13[a]	1.90 ± 0.67[a]	0.93 ± 0.26[b]	2.29 ± 0.95[a]	0.86 ± 0.30[a]
P- value	0.33	0.00	0.00	0.01	0.00

- Values are means ± S.D.
- Different letters refer to a significant difference (p≤ 0.05).
- Same letters refer to non a significant difference (p≤ 0.05).

Table (4) showed significant decrease (P≤0.05) in TC in first group compared with second group for patients, while there was non-significant in T.G, HDL in first and second group. There was significant decrease (P≤0.05) in LDL in first group compared with second group for patients. Also, there was non-significant in VLDL in first and second group.

Table (4) showed relationship between lipid profile in the patients group

Parameters / groups	TC (mmol/L)	T.G (mmol/L)	HDL (mmol/L)	LDL (mmol/L)	VLDL (mmol/L)
First group (Male patients)	3.76 ± 1.15 [b]	1.84 ± 0.80[a]	0.94 ± 0.29[a]	1.96 ± 0.90 [b]	0.83 ± 0.36[a]
Second group (Female patients)	4.48 ± 0.99[a]	1.96 ± 0.49[a]	0.91 ± 0.21[a]	2.66 ± 0.88[a]	0.89 ± 0.22[a]
P- value	0.00	0.41	0.57	0.00	0.40

- Values are means ± S.D.
- Different letters refer to a significant difference ($p \leq 0.05$).
- Same letters refer to non- significant differences ($p \leq 0.05$).

Discussion:

Major cardiovascular events are the leading cause of death globally. Extensive research has been done over the decades to understand the extent of severity and fatality of the disease in order to control its effects and prevent mortality [17].

The current study found that patients had a significant increase in troponin I and CK-MB, and these results were consistent with [18, 19, 20,21]. While it did not agree with study [22] that showed no significant change in the level of CK-MB in the MI group.

Given the compensatory mechanisms to maintain necessary myocardial functions, irreversible injury does not happen for an hour or more [23]. Therefore, the differences in skeletal mass are leading to sex differences in CK-MB concentrations [24] and is likely to explain higher levels in men. This is not the case with cardiac troponin, which has excellent tissue specificity, and hence discrepancies in reference limits are likely to reflect variations in cardiovascular physiology or the prevalence of subclinical pathology in men and women [25]. This may explain why elevation in serum creatinine occurred in patients with AMI in the presence of high levels of CK (one of most known serum markers of AMI) due to infarct cardiac muscle [26,27]

The results of the current study showed that MI patients suffer from lipid dysfunction in the blood, and these results are consistent with study [28] where dyslipidemia is involved in more than half of the cases of ischemic heart disease [29, 30].

Dyslipidemia is divided into two parts according to the causative agent. Primary, which are typically genetic and secondary, which are resulting from lifestyle or underlying conditions such as diabetes or hypothyroidism. The sedentary lifestyle combined with too much dietary consumption of saturated fat, cholesterol and trans fats are involved in the majority of dyslipidemia cases, particularly in developed countries. In addition to diabetes and hypothyroidism, other secondary causes involve alcohol overuse, liver disease, chronic kidney disease and some drugs, such as thiazides, beta-blockers, retinoids, highly active antiretroviral agents, estrogen and progestin [29].

The present study found that females had significantly higher levels of TC and LDL than males with no significant difference between the two genders in the other lipid parameters while these results

contrasted with a study [31]. The lipid profile must be assessed in every patient admitted with acute coronary syndrome within the first 24 hours and then periodically until a steady healthy state is achieved. The change within the first 24 hours is minimal, and then phasic changes follow. Hence, the first measurement can serve as a relatively reliable source to inform the selection of the lipid-lowering therapy. However, correctly recognizing the baseline is still difficult. Since a decreasing trend in TC, LDL, and HDL is seen periodically after MI, lipid-lowering therapy must be initiated even if the results are within the physiological range in the first few days [32].

There is a complex network of hormone action in combination with other, possibly sex-specific, direct or indirect modulators of lipid metabolism. These factors are responsible for sexual dimorphism in lipid metabolism. The precise nature of these factors is uncertain, but there are apparently many *e.g.* insulin and adipokines [33] that need to be explored in the future. There are clear differences in insulin action between men and women according to candidate. Particularly, there are differences between the sexes in insulin sensitivity of glucose metabolism in the liver and muscle. However, little is known concerning potential sex differences in the regulation of lipid metabolism via insulin [34].

In conclusion, there is negative relationship between lipids and myocardial infarction. Also, there is significant difference between male and female patients in TC and LDL. In patients admitted with myocardial infarction, serum lipid profile evaluation must be mandatory within the first 24 hours of admission.

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