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Association between Acute Myocardial Infarction, Lipid Profile and Smoking Habit

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Abstract:
Background: *Myocardial Infarction is the most common form of heart disease and the single most important cause of death in the developed and developing world. Myocardial infarction is the irreversible necrosis of heart muscle secondary to prolonged ischemia. The death of heart muscle from the sudden blockage of a coronary artery by a blood clot.*
Objective: *The aim of this study was to describe the association between total cholesterol, high density lipoprotein (HDL)-cholesterol, low density lipoprotein (LDL)-cholesterol and triglycerides in serum of patients with myocardial infarction. Also the association between smoking and development of myocardial infarction.*
Methods: *In this study hundred and seventy four patients (125 males and 49 females) of Libyan attended in an Emergency department and Intensive Care in Tajoura National Heart Center during the period from 4/2013 to 12/2013, with diagnosis of myocardial infarction and their age range from 30-90 years old.*
Results: *In the present study the results indicated that myocardial infarction is more common in male patients (71.8%) than female patients (28.2%). Males are much more liable to myocardial infarction than female patients in the age ranges less than 50 years old. However with age range of more than 60 years old myocardial infarction is more common in female patients than male patients. The results of the present study, showed that there is no clear evidence that increased total blood cholesterol and triglycerides concentration play an important role in development of myocardial infarction in the patients included in this study, if compared with the high percentage of patients with myocardial infarction and having normal lipid profile, concerning cholesterol and triglycerides blood level. Our study demonstrates that the percentage of smokers with myocardial infarction, is significantly higher than non smokers. The percentage of smoker male patients with myocardial infarction is (61.6) and that of non smoker male patients is (38.4).*
Conclusion: *No clear evidence that increased total blood cholesterol and triglycerides concentration play an important role in development of myocardial infarction in those patients included in this study. The effect of cigarette smoking on the development of myocardial infarction was more pronounced in younger age male patients than older ones.*

I. Introduction

Myocardial infarction is a term applied to myocardial necrosis secondary to an acute interruption of the coronary blood supply. Acute myocardial is considered, more appropriately part of a spectrum referred to as acute coronary syndromes, which also includes unstable angina and non–ST-elevation. Patients with ischemic discomfort may or may not have ST-segment elevation. Most of those with ST-segment elevation will develop Q waves. Those without ST elevations will ultimately be diagnosed with unstable angina based on the presence of cardiac enzymes[(1)]. Approximately 90% of myocardial infarction results from an acute thrombus that obstructs an atherosclerotic coronary artery. The interaction between LDL cholesterol and HDL-cholesterol indicated that risk is 11.6-fold higher for men with LDL cholesterol levels above 160 mg/dl and HDL-cholesterol levels below 35 mg/dl as compared to men with LDL cholesterol levels below 100 mg/dl and HDL-cholesterol levels above 45 mg/dl [(2)]. The relationship between HDL cholesterol and coronary heart disease is complex and comprises at least three independent issues. **First**, a low level of circulating high density lipoprotein cholesterol is a surrogate marker for an atherogenic metabolic situation commonly known as the metabolic syndrome, which also comprises the components obesity, hypertension, insulin resistance, and hypertriglyceridemia. **Second,** a low level of HDL cholesterol is a disease marker for advanced atherosclerosis which is related to its role as a negative acute phase reactant. **Third**, several strands of evidence indicate that at a low level of circulating high density lipoprotein may be causally related to the development of atherosclerosis[(3)]. Based on a Cox regression model, the incidence of myocardial infarction and mortality increased with increasing serum triglyceride values. The role of triglycerides as an independent risk factor for cardiovascular disease has been debated during a long time. High triglyceride levels are a significant independent predictor of coronary heart disease and ischemic stroke. Hypertriglyceridemia is often associated with low high-density lipoprotein cholesterol that contributes to cardiovascular disease and stroke. Hypertriglyceridemia may be associated with rheological and impaired fibrinolytic mechanisms that can

contribute to the development of atherothrombosis[4]. Statins, or 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors, have shown potent systemic antioxidant, antiinflammatory, and antiproliferative effects that are beyond their lipid-lowering effects and are relevant in the treatment of atherosclerosis-related diseases [5]. A number of additional drugs that interfere with vascular wall injury may play a role in slowing the progression of atherosclerosis. For example, aspirin has an important role in primary prevention of cardiovascular events. In addition to decreasing platelet activation, aspirin decreases the expression of inflammatory mediators such as inducible nitric oxide synthase, C-reactive protein, and interleukin-6 and tumor necrosis factor and inhibits vascular smooth muscle cell proliferation [6].

II. Material And Methods

Aliquots of 10 ml. of blood samples were obtained from one hundred and seventy four patients who were presented to Emergency department and intensive care unit in Tajoura National Heart Center, with a diagnosis of Myocardial Infarction during the period from 4/2013 to 12/2013; for determination of triglycerides, and cholesterol concentrations. The level of serum triglycerides were determined by GPO-PAP method as described by Fossatic and Principe[7]. However the level of serum cholesterol were determined by CHOD-PAP(Enzymatic colorimetric method) as described by Richmond[8].

III. Results & Discussion

The distribution of patients according to their age and sex were illustrated in **table (1),** where **5.6**% of males and **2**% of female patients were less than 40 years old; **23.2**% males and **4**% females having ages ranged from 40-50 years old. Age range 51-60 years old included **26.4**% of males and **30.6** % of females. However age range 61-70 included **17.6**% of males and **32.8** % of females. Finally **27.2**% of males and **30.6**% of females were more than 71 years old. These results indicated that myocardial infarction is more common in male patients 125(71.8%) than female patients 49 (28.2%) of all cases admitted to Tajoura National Heart Center during the period from 4/2013 to 12/2013. Males are much more liable to myocardial infarction (5.6% and 23.2%) than female patients (2% and 4%) in the age ranges less than 40 and 41-50 years old respectively. However with age range 61-70 years old myocardial infarction is more common in female patients (32.8%) than male patients (17.6%). This is followed by age range of 51-60; and age group older than 71 years, where the rate of myocardial infarction is more or less equal in both males and females. Myocardial infarction slightly more in female patients (30.6%) than male patients (26.4%) in patients with age range of 51-60 years. Also this was observed in patients with age older than 70 years, the rate of myocardial infarction is slightly more in female patients (30.6%) than male patients (27. 2%).

Table (1): Distribution of patients included in the study according to their age and sex.

Age / Sex	Less than 40 years		40-50 years		51-60 years		61-70 years		More than 70 years		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Male	7	5.6	29	23.2	33	26.4	22	17.6	34	27.2	125	100
Female	1	2	2	4	15	30.6	16	32.8	15	30.6	49	100
Total	8	4.6	31	17.8	48	27.6	38	21.8	49	28.2	174	100

N.B: % was correlated to the total number of patients in each group.

Epidemiologic data have indicated that young women are relatively spared from coronary heart disease**.** Although the reasons for this protection are not entirely clear, estrogen is thought to play a part. Women in whom suffering from myocardial infarction at young age may be predisposed to have particularly aggressive disease or possibly early onset or may have more risk factors for coronary heart disease, which might override the protective effect of estrogen. For example, diabetes has been found to negate the protective effect of female sex against coronary heart disease and death from cardiovascular disease [9].

The distribution of patients with myocardial infarction in the study according to their age and their lipid profiles were illustrated in tables (2, 3, 4 and 5)

Table (2): Incidence of myocardial infarction in male patients according to their lipid profile (cholesterol and triglycerides).

Age / State	Less than 40 years		40-50 years		51-60 years		61-70 years		More than 70 years		Total	
	No	%	No.	%	No.	%	No.	%	No.	%	No.	%
NBCC.	6	4.8	24	19.2	29	23.23	17	13.6	23	18.4	99	79.2
AHBCC.	1	0.8	5	4	4	3.2	5	4	11	8.8	26	20.8
NBTGC.	6	4.8	23	18.4	26	20.8	18	14.4	21	16.8	94	75.2
AHBTGC.	1	0.8	6	4.8	7	5.6	4	3.2	13	10.4	31	24.8

Table (3): Incidence of myocardial infarction in female patients according to their lipid profile (cholesterol and triglycerides).

Age / State	Less than 40 years		40-50 years		51-60 years		61-70 years		More than 70 years		Total	
	No	%	No.	%	No.	%	No.	%	No.	%	No.	%
NBCC.	1	2	2	4	9	18.4	14	28.6	12	24.5	38	77.6
AHBCC.	0	0	0	0	6	12.2	2	4	3	6.1	11	22.4
NBTGC.	1	2	2	4	10	20.4	13	26.5	14	28.6	40	81.6
AHBTGC	0	0	0	0	5	10.2	3	6.1	1	2	9	18.4

N.B: 1-% was correlated to the total number of female patients included in the study(49).
2- NBCC. = Normal blood cholesterol concentration.
3- AHBCC. = Abnormal high blood cholesterol concentration.
4- NBTGC. Normal blood triglycerides concentration..
5- AHBTGC.= Abnormal high blood triglycerides concentration.

The results indicated that in case of male patients the percentages of those having hypercholesterolemia and hypertriglyceridemia were **20.8**% and **24.8** respectively. However in case of female patients the percentages of those having hypercholesterolemia and hypertriglyceridemia were **22.4** % and **18.4** % respectively. Also in case of male patients the percentage of those having abnormal high concentration of low density lipoprotein cholesterol was **20.8 %** and **26.5%** in case of female patients. While the percentages of abnormal low concentrations of high density lipoprotein cholesterol were **28%** for male patients and **34.7%** for female patients. This means that there is no clear evidence that increased total blood cholesterol and triglycerides concentration play an important role in development of myocardial infarction in the patients included in this study, if compared with the high percentage of patients with myocardial infarction and having normal lipid profile, concerning cholesterol and triglycerides blood level. This means that serum cholesterol and triglycerides levels do not discriminate well between individuals with and without myocardial infarction. This finding was in agreement with the result concluded that heart attack has been reported to present in normocholesterolemic subjects or hypercholesterolemic ones [(10)]. In another study that considered coronary heart disease prediction using total cholesterol (TC), LDL cholesterol (LDL-C), TC/HDL-C ratio, and LDL-C/HDL-C ratio, concluded that "total cholesterol/HDL is a superior measure of risk for coronary heart disease compared with either total cholesterol or LDL cholesterol, and that current practice guidelines could be more efficient if risk stratification was based on this ratio rather than primarily on the LDL cholesterol level." Such an approach appears attractive, but at the extremes of the TC or LDL-C distribution, equal ratios may not signify the same coronary heart disease risk. Moreover, use of a ratio may make it harder for the physician to focus on the separate values for TC, LDL-C, and HDL-C that have to be borne in mind to make appropriate clinical decisions concerning therapy [(11-15)].

The incidence of myocardial infarction in cigarette smoker and non smoker patients was illustrated in table (6). The results indicated that the percentage of smokers with myocardial infarction, is significantly higher than non smokers. The percentage of smoker male patients with myocardial infarction is (61.6) and that of non smoker male patients is (38.4). However the effect of cigarette smoking on the development of myocardial infarction was more pronounced in younger age male patients than older ones. The percentage of smokers with myocardial infarction in age ranges, (40-50), (51-60) and (more than 70) years; were (15.2%), (16.8%) and (16%) respectively. Meanwhile The percentage of smokers with myocardial infarction in age range (61-70), was (9.6%). This indicates the most important relation between smoking and myocardial infarction development, especially with male patients. Tobacco use is heavily influenced by the historical context of communities and cultural norms, and in most societies women have smoked less than men especially in Arab communities where the majority of females are non smokers. Tobacco use clearly explains part of the lower rate of myocardial infarction in younger women compared to that in men. Our study demonstrates that the percentage of smokers with myocardial infarction, is significantly higher than non smokers. The percentage of smoker male patients with myocardial infarction is (61.6) and that of non smoker male patients is (38.4). However the effect of cigarette smoking on the development of myocardial infarction was more pronounced in younger age male patients than older ones.

Table (4): Incidence of myocardial infarction in male patients according to their lipid profile(LDL and HDL cholesterol concentrations).

Age	Less than 40 years		40-50 years		51-60 years		61-70 years		More than 70 years		Total	
State	No	%	No.	%	No.	%	No.	%	No.	%	No.	%
NLDLC.	6	4.8	23	18.4	29	23.23	18	14.4	23	18.4	99	79.2
AHLDLC.	1	0.8	6	4.8	4	3.2	4	3.2	11	8.8	26	20.8
NHDLC.	6	4.8	20	16	26	20.8	16	12.8	22	17.6	90	72
ALHDLC.	1	0.8	9	7.2	7	5.6	6	4.8	12	9.6	35	28

Table (5): Incidence of myocardial infarction in female patients according to their lipid profile (LDL and HDL cholesterol concentrations).

Age	Less than 40 years		40-50 years		51-60 years		61-70 years		More than 70 years		Total	
State	No	%	No.	%	No.	%	No.	%	No.	%	No.	%
NLDLC.	1	2	2	4	10	20.4	12	24.5	11	22.4	36	73.5
AHLDLC.	0	0	0	0	5	10.2	4	3.2	4	3.2	13	26.5
NHDLC.	1	2	2	4	9	18.4	10	20.4	10	20.4	32	65.3
ALHDLC.	0	0	0	0	6	4.8	6	4.8	5	10.2	17	34.7

N.B: 1-% was correlated to the total number of female patients included in the study(49).
2- NLDLC.= Normal concentration of low density lipoprotein cholesterol.
3- AHLDLC.= Abnormal high concentration of low density lipoprotein cholesterol.
4- NHDLC.= Normal concentration of high density lipoprotein cholesterol.
5- ALHDLC.= Abnormal low concentration of high density lipoprotein cholesterol.

Table (6): Incidence of myocardial infarction in male patients according to smoking habit.

Age	Less than 40 years		40-50 years		51-60 years		61-70 years		More than 70 years		Total	
Smoking Habit	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Smokers	5	4	19	15.2	21	16.8	12	9.6	20	16	77	61.6
Non smokers	2	1.6	10	8	12	9.6	10	8	14	11.2	48	38.4
Total	7	5.6	29	23.2	33	26.4	22	17.6	34	27.2	125	100

N.B: % was correlated to the total number of male patients (125) included in the study.

Epidemiological data have consistently shown that the risk of myocardial infarction increases progressively with the number of cigarettes smoked per day. Studies have demonstrated that the risk of fatal and nonfatal myocardial infarction is about two to three times higher in smokers than in nonsmokers, and the risk of sudden cardiac death may be 10 times higher. Furthermore, continued smoking after myocardial infarction may double the rate of reinfarction and subsequent death [(16)].

Elevated hematocrit, fibrinogen, and platelet levels suggest that smokers may have a hypercoagulable state promoting coronary thrombosis. Also Clinical studies have demonstrated that cigarette smoking constricts both epicardial coronary arteries and myocardial resistance vessels and thus may contribute to infarction [(17)].

Smoking was found to influence risk significantly in a dose-dependent manner, the risk increasing 2% to 3% for each gram of tobacco smoked daily. Risk was particularly associated with inhalation, the risk for inhalers being almost twice that of non inhalers. No difference in risk could be demonstrated between various types of tobacco (pipe, cigar/cheroots, or plain and filtered cigarettes). The risk seemed associated with current smoking only, in as much as the duration of the smoking habit was not important. Ex-smokers had the same risk as those who had never smoked regardless of duration of smoking and time elapsed since quitting. Relative excess risk was significantly higher in female smokers than in male smokers, and daily alcohol intake appeared to have some protective effect on the risk of first acute myocardial infarction [(18-20)].

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