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Serum lipid profiles in acute myocardial infarction patients in Gorgan

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Abstract

Acute myocardial infarction is one of the important reasons of death and unhealthiness in the world. The present study was undertaken to investigate the changes in serum lipids and lipoproteins in patients with acute myocardial infarction. This study was performed in the Biochemistry and Metabolic Disorder Research Center of Gorgan, Golestan province (South East of Caspian Sea), Iran in 2011.The levels of lipid profile were significantly changed in the acute myocardial infarction patients. acute myocardial infarction patients had significantly higher levels of total cholesterol, LDL-cholesterol, VLDL-cholesterol, TG, LDL-cholesterol /HDL-cholesterol, total cholesterol /HDL-cholesterol, LDH, CPK and CPK-MB and lower level of HDL-cholesterol, as compared to the control subjects. We found a significant association of lipid profiles with acute myocardial infarction. Changing of dietary and social activity habits of people in this area can help to prevent future atherogenic damaging in AMI patients. Changing of dietary and social activity habits of people in this area can help to prevent future atherogenic damaging in AMI patients. Reduced serum HDL-cholesterol in our study subjects may be one of the effective reasonable lipid disorders in AMI patients.

Key words: lipid profiles, acute myocardial infarction, Gorgan

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Introduction

Acute myocardial infarction is one of the important reasons of death and unhealthiness in the world. Various risk factors for coronary heart disease have been reported, including age, gender, race and family history and an another risk factors, like serum cholesterol, smoking, diabetes and high blood pressure can be changed [1**]**. Myocardial Infarction (MI) comes together as a results of environmental causes and personal susceptibilities [2-5]. In the United States and United Kingdom, nearly 650,000 and 180000 patients get an acute myocardial infarction (AMI) every year**,** respectively [6**]**. According to WHO planned in the year 2005 that the coronary heart disease causes for 7.6 million deaths (13.2% of total deaths) whole world. Above 80% of these deaths took place in fewer developing countries [7]. In our country, Iran, rate of death because of cardiovascular disease comprised 26.6% and 47.3% of deaths in the years 1987 and 1995, respectively [8]. Acute myocardial infarction accounts for a very many of deaths in Iran [9]. There are several assessments on the variations of serum lipid profiles following myocardial infarction. Many researchers have reported a decrease in total cholesterol [10-19], HDL-cholesterol [18] and LDL-cholesterol [16-18] after acute myocardial infarction. In another studies showed that there is no modification in serum total cholesterol [20], HDL-cholesterol [16, 20] and triglycerides levels [13, 20-23]. Various studies have shown that the total cholesterol to HDL cholesterol and of LDL cholesterol to HDL cholesterol ratios can be used as predictors of acute coronary occurrences [24-25]. The present study was undertaken to investigate the changes in serum lipids and lipoproteins in Patients with acute myocardial infarction in Gorgan (South East of Caspian Sea), Iran.

Materials and Methods

This study was performed in the Biochemistry and Metabolic Disorder Research Center of Gorgan, Golestan province (South East of Caspian Sea), Iran in 2011. The study group included 35 subjects (12 females and 23 males) with acute myocardial infarction (with the mean age of 57.22±11.44 years old) who were referred to the intensive Coronary Care Unit in 5th Azar Hospital (the only general teaching hospital in Gorgan), Gorgan Faculty of Medicine, Golestan University of Medical Sciences. None of the patients had thyroid dysfunction, diabetes, hypertension and liver or kidney disease. All the included subjects provided an informed consent. We also chosen 35 healthy subjects (12 females and 23 males with

the mean age of 55.80±13.15 years old) as our control group and matched them for age and gender. The exclusion criterion was the coexistence of any other serious illness. A venous blood sample was collected from all the subjects after 12-hours overnight fast. The samples were centrifuged for 10 minutes at 3000 rpm. Fasting levels of serum blood glucose, total cholesterol, High Density Lipoprotein (HDL) cholesterol, Low Density Lipoprotein cholesterol (LDL) Very Low Density Lipoprotein cholesterol (VLDL), Triglycerides (TG), lactate dehydrogenase (LDH) and Creatine Phospho Kinase (CPK) and Creatine Phospho Kinase-MB (CPK-MB) levels were measured by enzymatic methods using spectrophotometer techniques (Model JENWAY 6105 UV / VIS) in the Biochemistry and Metabolic Disorder Research Center (Faculty of Medicine). Acute myocardial infarction was defined as prolonged chest pain and was diagnosed as acute myocardial infarction on clinical examination, electrocardiographic changes and laboratory basis. The results were reported as mean ± SD. The statistical analysis was done with SPSS- 11.5 version software. The results were evaluated by using student't' test to determine the significance of biochemical parameters among two groups. Statistical significance was considered at $P < 0.05$.

Results

The clinical characteristics of acute myocardial infarction patients and control subjects have been shown in tables 1, 2, 3, 4 and 5. A number of obvious differences between two subjects are found. Table 1 shows that acute myocardial infarction patients had higher levels of total cholesterol, LDL-cholesterol, VLDL-cholesterol, TG, LDL-cholesterol /HDL-cholesterol, total cholesterol /HDL-cholesterol, LDH, CPK and CPK-MB and lower level of HDL-cholesterol, as compared to the control subjects. Notably, all above mentioned biochemical parameters (HDL-cholesterol was significantly lower) were significantly higher in acute myocardial infarction patients than in control subjects ($P< 0.05$) (Table1).Table 2 shows comparison of biochemical parameters between males and females acute myocardial infarction patients. The data shown in Table 2 reveals that there was no significant difference in all parameters in female and male patients. Table 3 shows comparison of biochemical parameters between males and females control groups. There was no significant difference in all parameters in both genders. The data of Table 4 shows that female acute myocardial infarction patients had significantly higher levels of total cholesterol, LDL-cholesterol, VLDL-cholesterol, TG, LDL-cholesterol /HDL-cholesterol, total cholesterol /HDL-cholesterol LDH, CPK and CPK-MB and significantly lower level of HDL-cholesterol, as compared to female control subjects ($P< 0.05$). The data of Table 5 shows that male acute myocardial infarction patients had significantly higher levels of in table 4 mentioned biochemical parameters when compared with male control subjects ($P< 0.05$). But this is not the same for total cholesterol in male acute myocardial infarction patients when compared with male control subjects

Table 1. *Biochemical parameters in acute myocardial infarction patients and control groups*

Parameters	Acute myocardial infarction patients (n=35)	Control groups (n=35)	P-value
Age (year)	**57.22±11.44**	55.80±13.15	0.605
Fasting blood sugar (mg/dl)	**108.43**±15.81	99.94±8.26	0.013
Blood urea nitrogen (mg/dl)	**20.60**±8.34	18.97±3.75	0.332
Creatinine (mg/dl)	**1.08**±0.23	0.99±0.14	0.057
Creatine phosphokinase (U/L)	2104.10±1744.73	123.63±27.17	0.001
Creatine phosphokinase MB(U/L)	**165.09**±135.52	22.11±3.45	0.001
Low density lipoprotein (mg/dl)	**117.03**±22.67	87.57±20.78	0.001
High density lipoprotein (mg/dl)	**40.62**±5.18	61.57±11.59	0.001
Very Low density lipoprotein (mg/dl)	33.73±3.46	24.73±6.34	0.001
Total cholesterol(mg/dl)	**201.86**±44.24	181.37±15.32	0.001
Triglycerides (mg/dl)	168.69±17.34	123.69±31.71	0.001
Lactate dehydrogenises(U/L)	**1590.20**±786.34	369.83±53.73	0.001
LDL-cholesterol/HDL-cholesterol	2.95±0.80	1.41±0.46	0.001
Total cholesterol/ HDL-cholesterol	5.08±1.46	3.03±0.59	0.001

Table 2: *Comparison of biochemical parameters between males and females acute myocardial infarctionPatients*

Parameters	Females (n=12)	Males (n=23)	P-value
Age (year)	60.33±8.27	55.60±12.65	0.329
Fasting blood sugar (mg/dl)	109.17±12.93	108.04±17.39	0.693
Blood urea nitrogen (mg/dl)	20.58±4.85	20.60±9.79	0.423
Creatinine (mg/dl)	1.10±0.26	1.06±0.22	0.845
Creatine phosphokinase(U/L)	1843.80±566.43	2239.90±2118.46	0.394
Creatine phosphokinase MB(U/L)	137.25±119.03	179.61±143.72	0.429
Low density lipoprotein (mg/dl)	119.50±25.84	115.74±21.34	0.249
High density lipoprotein (mg/dl)	42.50±5.77	39.65±4.68	0.178
Very Low density lipoprotein (mg/dl)	34.33±3.81	33.42±3.32	0.566
Total cholesterol(mg/dl)	217.17±57.20	193.87±34.57	0.169
Triglycerides (mg/dl)	171.67±19.05	167.13±16.60	0.566
Lactate dehydrogenises(U/L)	1474.20±766.0	2101.70±809.78	0.055
LDL-cholesterol/HDL-cholesterol	2.91±0.98	2.97±0.71	0.841
Total cholesterol/ HDL-cholesterol	5.30±2.02	4.96±1.11	0.682

Table 3. *Comparison of biochemical parameters between males and females control subjects*

Parameters	Females (n=12)	Males (n=23)	P-value
Age (year)	58.75±15.19	54.25±12.03	0.864
Fasting blood sugar (mg/dl)	101.42±8.10	99.17±8.42	0.125
Blood urea nitrogen (mg/dl)	17.91±4.39	19.52±3.34	0.786
Creatinine (mg/dl)	0.98±0.12	0.99±0.15	0.590
Creatine phosphokinase (U/L)	119.42±27.26	125.83±27.47	0.273
Creatine phosphokinase MB (U/L)	21.08±2.60	22.65±3.76	0.213
Low density lipoprotein (mg/dl)	88.41±8.08	87.13±25.18	0.782
High density lipoprotein (mg/dl)	55.91±8.42	64.52±12.05	0.365
Very Low density lipoprotein (mg/dl)	23.90±9.47	25.17±4.09	0.930
Total cholesterol(mg/dl)	182.0±19.82	181.04±12.88	0.703
Triglycerides (mg/dl)	119.50±47.35	125.87±20.46	0.930
Lactate dehydrogenises (U/L)	375.67±51.44	366.78±55.76	
LDL-cholesterol/HDL-cholesterol	1.60±0.22	1.42±0.54	0.369
Total cholesterol/ HDL-cholesterol	3.29±0.51	2.89±0.60	0.406

Table 4. *Comparison of biochemical parameters between females acute myocardial infarction patients and control subjects*

Parameters	Females acute myocardial infarction patients (n=12)	Females control subjects (n=12)	P-value
Age (year)	60.33±8.27	58.75±15.19	0.703
Fasting blood sugar (mg/dl)	109.17±12.93	101.42±8.10	0.065
Blood urea nitrogen (mg/dl)	20.58±4.85	17.91±4.39	0.148
Creatinine (mg/dl)	1.10±0.26	0.98±0.12	0.192
Creatine phosphokinase(U/L)	1843.80±566.43	119.42±27.26	0.001
Creatine phosphokinase MB(U/L)	137.25±119.03	21.08±2.60	0.001
Low density lipoprotein (mg/dl)	119.50±25.84	88.41±8.08	0.003
High density lipoprotein (mg/dl)	42.50±5.77	55.91±8.42	0.001
Very Low density lipoprotein (mg/dl)	34.33±3.81	23.90±9.47	0.001
Total cholesterol(mg/dl)	217.17±57.20	182.0±19.82	0.043
Triglycerides (mg/dl)	171.67±19.05	119.50±47.35	0.012
Lactate dehydrogenises(U/L)	1474.20±766.0	375.67±51.44	0.001
LDL-cholesterol/HDL-cholesterol	2.91±0.98	1.60±0.22	0.001
Total cholesterol/ HDL-cholesterol	5.30±2.02	3.29±0.51	0.004

Table 5. *Comparison of biochemical parameters between males acute myocardial infarction patients and control subjects*

Parameters	**Males acute myocardial infarction patients** (n=23)	**Males control subjects** (n=23)	**P-value**
Age (year)	55.60±12.65	54.25±12.03	0.715
Fasting blood sugar (mg/dl)	108.04±17.39	99.17±8.42	0.066
Blood urea nitrogen (mg/dl)	20.60±9.79	19.52±3.34	0.652
Creatinine (mg/dl)	1.06±0.22	0.99±0.15	0.117
Creatine phosphokinase (U/L)	2239.90±2118.46	125.83±27.47	0.001
Creatine phosphokinase MB (U/L)	179.61±143.72	22.65±3.76	0.001
Low density lipoprotein (mg/dl)	115.74±21.34	87.13±25.18	0.001
High density lipoprotein (mg/dl)	39.65±4.68	64.52±12.05	0.001
Very Low density lipoprotein (mg/dl)	33.42±3.32	25.17±4.09	0.001
Total cholesterol(mg/dl)	193.87±34.57	181.04±12.88	0.114
Triglycerides (mg/dl)	167.13±16.60	125.87±20.46	0.001
Lactate dehydrogenises (U/L)	1650.0±806.89	366.78±55.76	0.001
LDL-cholesterol/HDL-cholesterol	2.97±0.71	1.42±0.54	0.001
Total cholesterol/ HDL-cholesterol	4.96±1.11	2.89±0.60	0.001

Discussion

Coronary heart diseases are the most important factor of death [26]. There is an increasing prevalence of these diseases in developing countries [7]. Study of Bitla et al **[**27] showed that Patients with MI showed no significant change in lipid profile when compared to healthy controls. Ryder et al. [28] reported that there is no significant alteration in triglyceride levels. Study of Vetter et al. [29] showed that triglyceride levels diminished slowly from the second hour after myocardial infarction. Some studies showed that there are correlations between the occurrence of AMI and abnormality of lipid profiles [30]. Some other study showed that there was an increase in serum triglycerides during AMI [31, 32]. Study of Salahuddin et al. revealed significantly high levels of triglycerides and low levels of HDL-cholesterol in AMI patients compared to control subjects [32]. In our present study, serum triglyceride levels showed significant increase in AMI patients when compared with control subjects. According to the present study, this result was in agreement with our finding. There is a different mechanism about elevation of triglycerides after MI. It is reported that elevated triglyceride levels may depend on genetic basis [33] and nutritional habits [34]. Triglyceride levels may change because of inherited abnormality of very low density lipoprotein It may be happen because of increased flowing of fatty acids and impaired elimination of VLDL from the plasma [35].We found significant change in serum total cholesterol levels after acute myocardial infarction. Our study is in agreement and in contrast to that by other researchers who found either an increase [36, 12] or a decrease [37-39, 28 and 40] or normal cholesterol [30, 41] during the acute myocardial infarction. This shows that serum cholesterol level reveal no differences between persons with and without AMI. Elevated serum cholesterol has depended on elevated consumptions of fat [42] and genetic basis [33]. LDL carries the most of cholesterol in the plasma and increasing of LDL depend on increasing of total cholesterol level [43]. Reduction of HDLc concentration in our findings is not in agreement with the other studies that shown either a increase [40] or no change [41] and in agreement with the other study [32]. Several studies have supported that the ratios of LDL-cholesterol /HDL-cholesterol and total cholesterol /HDL-cholesterol show the atherosclerotic injury of the wall of the vessels [44-46]. We found significant increase in ratios of LDL-cholesterol /HDL-cholesterol and total cholesterol /HDL-cholesterol levels after acute myocardial infarction. The latest studies in agreement with our studies. Study of Some studies have shown that there is a significant decrease in LDL-cholesterol following MI [47, 28]. Sandkamp et. al. (48) showed that LDL-Ch level increased and this is in agreement with our finding. LDL-Ch level may be increase as a result of reducing in refining or excessive production of LDL-Ch [49]. LDL cell surface receptors cleaned LDL-Ch from the circulation. These receptors may change as a result of coronary heart disease, thus uptake of LDL-Ch is decreased [50]. On the other hand, increasing of LDL possibly as a result of excessive production of VLDL and /or decreasing of VLDL remnants (Low activity of LDL receptors)[51]. The results of our study shows significant increase in total cholesterol, LDL-cholesterol, VLDL-cholesterol, TG, LDL-cholesterol /HDL-cholesterol and total cholesterol /HDL-cholesterol after acute myocardial infarction. Thus, we found a significant association of lipid profiles with acute myocardial infarction. Changing of dietary and social activity habits of people in this area can help to prevent future atherogenic damaging in AMI patients. Reduced se-

rum HDL-cholesterol in our study subjects may be one of the effective reasonable lipid disorders in AMI patients.

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