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Monitoring Lipid profile in patients with acute myocardial infarction at King Hussein medical

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Abstract

Lipid profile is a panel of blood tests that serves as an initial broad medical screening tool for abnormalities in lipids such as cholesterol, triglycerides, HDL&LDL.

The results of these tests can identify certain genetic diseases and determine approximate risks for cardiovascular disease.
The aim of this study is to monitor lipid profile in patients with acute myocardial infarction at King Hussein medical.

Serum lipids were estimated in 30 patients with acute myocardial infarction during acute phase (day 1, 2, 3), predischarge and after three months. Serum triglycerides, total cholesterol (TC) and LDL-cholesterol (LDLc) showed no significant change during the hospital stay and three months follow up. HDL-cholesterol (HDLc), however, started falling from day 2 onwards with statistically significant reduction at pre-discharge and remained so at 3 months. The ratios of TC/HDLc and LDLc/HDLc showed significant increase on predischarge day as compared to day 1.

Serum triglycerides also showed an increasing trend after myocardial infarction with a significant increase on day 3 and predischarge as compared to day 1. It is concluded that the optimum time for assessment of serum lipid profile in patients with myocardial infarction seems to be within 24 hours of the acute episode.

KEY WORDS: Lipids, Cholesterol, triglycerides, Myocardial Infarction

INTRODUCTION

Opinion is divided on the changes that occur in serum lipids and lipoproteins following myocardial infarction (MI). Most workers have reported a reduction in total cholesterol, HDL-cholesterol and LDL cholesterol after acute MI. Others have, however, reported no change in serum total cholesterol and HDL-cholesterol.

Similar variations have also been noted in serum triglycerides levels**.**

From these reports it is clear that phasic changes do occur in patients following MI and therefore there is a recommendation for detection of hyperlipidaemia in patients with acute MI that the serum lipids should be assessed either within 24 hours after infarction or after 2-3 months of acute MI.While the recommendation may hold true for absolute levels there is no consensus on when ratios of various fractions of lipids should be assessed.

The present study was, therefore, undertaken to examine the changes in serum lipids including ratios of total cholesterol/HDL-cholesterol and LDL-cholesterol/ HDL- cholesterol(1) .

MATERIALS AND METHODS

The study was carried out in 30 patients (24 male and 6 female) aged between 40 to 70 years, admitted to our intensive coronary care unit with acute myocardial infarction. The diagnosis of MI was established by clinical, ECG and serum cardiac enzymes examination. None of the patients had thyroid dysfunction, liver or kidney disease. Fasting blood samples were taken as soon as possible after admission (day 1, average 14 hours after chest pain), 24 hours later (day 2), 48 hours later (day 3), pre discharge (average day 7) and after 3 months. The dietary and life style changes where noted at 3 months follow up. Only those patients where finally included who were not taking any hypolipidemic drug. Serum total cholesterol, HDL-cholesterol and triglycerides were measured by readymade kits using enzymatic methods. LDL-cholesterol was calculated by Friedewald equation.

RESULTS AND DISCUSSION

The results of the present study have been shown in table 1. We found no significant change in serum total cholesterol levels either during the acute phase or after 3 months of MI.

Our study provides information which is in direct contrast to that by others who found either a decrease or an increase during the acute phase of MI. Although there was a decline in total cholesterol level on day 3 but it failed to reach statistical significance. Moreover, the pattern was almost same in males and females. Considering the pattern in individual patients the TC either did not change or decreased a bit (about 10%) on day 3. HDL-cholesterol levels, in our study, started falling from day 2 onwards, the reduction achieving statistical significance in pre-discharge specimens ($p<0.001$) and remained so at 3 months follow up ($p<0.01$) as compared to day 1. Ryder *et al.* (2) have also reported a significant reduction in HDL-cholesterol on predischarge day but in their study it tended to normalize after 3 months. In contrast to our findings of reduction in HDLc concentration other studies have shown either a rise (4) or no change (5) in HDLc. An

inverse relationship between TG and HDLc has been reported (6) and in our subjects too, this appears true. However, the low levels of HDLc at 3 months in our study indicate an irreversible decrement rather than an acute reversible change and since there were no preinfarction values of HDLc known, the effect of infarction can only be assumed and by no means proven (5). LDL cholesterol recorded no significant change throughout the study period 1(Table 1) in our subjects. However, a significant decrease in LDL-cholesterol following MI has been reported by others (2,3). Several studies have advocated the value of ratios of LDLc/HDLc and TC/HDLc as a correlate of the severity and extent of coronary artery stenosis (9). The present study showed an increase in the ratios of TC/HDLc and LDLc/HDLc on predischarge day as compared to day 1($p<0.01$ and <0.05 respectively). This was due to significant decrease in HDLc on predischarge day.

Serum triglyceddes showed an increasing trend after MI with a significant increase on day 3($p<0.01$) and predischarge ($p<0.001$) as compared to day 1. The values at 3 months were higher than day 1 but the difference was not statistically significant. This finding was in accordance with those by others(3,5). On the contrary, Vetter *et al.* (8) recorded a progressive fall in triglycerides levels from the second hour after MI and Ryder *et al.* (2) found no significant change in triglycerides. The mechanism of increase in triglycerides after MI may be due to elevated flux of fatty acids and impaired removal of VLDL from the plasma (7). Another possible mechanism for elevated TG levels may be the effect of 13-blockers but this contention seems to be invalid for increased TG levels on day 3 and pre-discharge (day 7) as β-blockers take about 2 weeks to show their effect on serum lipids (6). Moreover, the TG levels came down to near day 1 values even in patients takingβ-blockers. However, the normalization of TG levels at 3 months nearing day 1 levels could be due to diet restrictions and changes in overall lifestyle of the patients.Thus, our study reveals some significant alterations in serum HDL- cholesterol, triglycerides and ratios of TC/HDLc and LDLclHDLc after MI.

Table 1 : Serum lipids profile in patients with acute myocardial infraction

schedule	Total cholesterol mg/dl (TC)	HDL mg/dl	LDL mg/dl	Triglycerides mg/dl	Tc /HDL	LDL/HDL
Day 1	188 ±37	42 ±7.5	123.6±40	115±40	4.5±1.7	2.9±1.2
Day 2	187.7±40	41.2±7.3	120±8	140±38	4.6±1.5	2.8±1.2
Day 3	175.3±39	39.1±7.2	110±29	165±50	5.6±1.6	3.6±1.3
Pre discharge	189.2±35	34.1±7.5	123±30	170±52	5.9±1.2	3.3±1.2
3 months	182.4±37	36.1±5.9	121±8	135±44	5.3±1.3	3.5±1.1
	n=30	n=30	n=30	n=30	n=30	n=30
Values are mean±SD Significance of difference as compared to day 1: $p<0.05$,$p<0.01$,$p<0.001$						

The mechanism of these changes is still not clear. Could it be a metabolic effect of stress, hormones etc.? One recent study has shown that acute myocardial infarction causes a profound up regulation of cholesterol synthesis as acute phase response and the observed decrease of plasma cholesterol levels after acute myocardial infarction must, therefore, be explained by the parallel increase of LDL receptor activity and thus increased cholesterol catabolism (10). The mechanistic aspect of these changes deserves further investigations with larger number of patients.

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