

Serum Lipids and Lipoproteins in Non-Diabetic, and Non-Hypertensive Patients of IHD

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ABSTRACT

Background: Atherosclerosis is the most common cause of two main presentations of CAD i.e. angina pectoris (AP) and myocardial infarction (MI). Plasma lipids and lipoproteins abnormalities are frequently associated with the presence of atherosclerosis CAD.

Aim: To evaluate the role of lipids and complete lipoprotein profile in patients of angina pectoris and myocardial infarction with atherosclerosis.

Methods: A total of 109 male patients of CAD (AP 42 + MI 67) and 50 healthy control subjects were included in the study.

Results: This study found highly significant ($P < 0.001$) raised levels of total cholesterol except in AP, triglycerides, LDL, VLDL and significantly low levels of total HDL cholesterol and MI, when compared with controls. However, only significantly ($P < 0.02$) raised levels discriminated the patients of MI when compared with AP patients.

Conclusion: It is concluded that all lipids and lipoproteins play important direct or indirect role in atherosclerosis CAD.

Keywords: Atherosclerosis, myocardial infarction, Plasma lipids.

INTRODUCTION

The high mortality is common to Gujrati, Hindus, Punjabi Sikhs and Muslims from Pakistan and Bangladesh. The clinical picture in South Asian patients with CAD is similar to that in Europeans¹. Among the four major risk factors which lead to the development of atherosclerosis, the best documented one is the correlation between blood lipids and lipoproteins with CAD². Lipoprotein(a) is an LDL-like particle in which apo(a), a glycoprotein consisting of several repetitive kringle structures, is attached to apolipoprotein B by a disulphide linkage.³ Elevated concentration of Lp (a) has been identified by a meta-analysis as a modest risk factor for IHD in the general population. There are contradictory reports in the literature in terms of Lp(a) concentration among T2DM subjects, some studies reported lower⁴, others reporting higher, concentration compared to non-diabetic subjects.^{5,6} In terms of its relationship with IHD, again controversy exists in the literature⁷.

Variations in Lp(a) levels are mainly genetically determined⁸. Other factors like the diet, drugs, hormones and glycemic control were also found to effect Lp(a) concentration⁹. Lp(a) has also been found to be influenced by other lipid parameters an inverse relation was revealed between Lp(a) and

triglycerides and positive correlation with total and LDL cholesterol among diabetic subjects¹⁰. Therefore, this study was undertaken to investigate the level of Lp(a) as well as its relationship with other lipid parameters in a cohort of type 2 diabetic subjects with and without IHD.

SUBJECTS AND METHODS

One hundred and nine male patients of CAD (AP 42 +MI 67) and 50 healthy control subjects were included in the study. The data was analyzed by applying students 't' test for group comparison. The relationship and inter-relationship between two variables were analyzed by calculating correlation coefficient 'r' and regression lines respectively.

RESULTS

Lipids and lipoproteins values (mean±sem) obtained from control subjects, patients of AP and MI are arranged. The data showed that statistically significant ($P < 0.001$) difference in ratios of LDL/T-HDL-C and LDL-HDL2-C in patients of MI group, when compared with AP group (Tables 1-2).

Table 1: Comparison of serum values of lipids and lipoproteins

Ratio	AP (42)	MI (67)
LDL/T-HDL-C	3.77±0.26	5.31±0.3*
LDL/HDL2-C	13.7±1.35	23.33±0.12*

* $P < 0.001$ (Significant)

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Table 2: Comparison of serum values of lipids and lipoproteins

Serum (mg/dl)	Control (50)	A (42)	M (67)
Total cholesterol	195.58± 2.98	211.97± 6.53	225.55± 8.33*** ^B
Triglycerides	123.64± 4.12	190.16± 11.35*** ^A	199.70± 9.43*** ^B
LDL	116.08± 3.11	143.09± 8.195*** ^A	165.65± * ^C 8.90*** ^B
VLDL	53.92± 2.95	111.88± 8.62*** ^A	116.70± 743*** ^B
T-HDL-C	53.92± 1.10	34.73± 0.81*** ^A	32.10± ** ^C 0.63*** ^A
HDL3-C	29.10± 0.96	23.66± 0.74*** ^A	24.02± 0.52*** ^B
HDL2-C	24.18± 0.58	11.19± 0.75*** ^A	8.07± 0.35*** ^{BC}

*P<0.02, ** P<0.01, ***P<0.001

A. Control vs AP, B. Control vs MI C. AP vs MI

DISCUSSION

Comparative analysis of lipids and lipoproteins between AP and MI patients in this study showed significant difference at the level of LDL, T-HDL-C and HDL2-C hence difference is actually at LDL and HDL2-C level. Clinic-pathologic difference between AP and MI is primarily based on extent of narrowing of lumen of coronary vessels by atherosclerotic plaque¹¹. Cholesterol as a normal metabolite is not harmful unless it accumulates in the wrong place for example within wall of artery, from where it cannot be readily mobilized and its progression eventually leads to the development of atherosclerosis¹². The cholesterol that accumulates in atherosclerotic lesions, originate primarily from plasma proteins including LDL¹³. LDL particles are taken up by arterial wall¹⁴ and LDL receptor account for about 70% of total cholesterol flux¹⁵. Presently the main function attributed to HDL is removal of cholesterol from tissues and return to the liver. Entry of abnormal cholesterol concentration to and/or inefficient removal from tissues may lead to initiation or progression of atherosclerosis. Triglycerides and cholesterol are transported through lipoprotein in the blood. Metabolism of VLDL, LDL, HDL and chylomicron is closely linked with each other¹⁶.

The present study revealed that Lp(a) concentration is an independent risk factor for IHD among Omani T2DM subjects, irrespective of genders. Nevertheless, the actual concentration of Lp(a) had not reached statistical significance because of a number of limiting factors. Firstly; metabolic due to the accelerated catabolism of apo

(a)-TRPs particles. Secondly, gender influence, having subdivided the studied groups by gender because of its confounding influence, the number of subjects within each subgroup became smaller and therefore, might have influenced the power of the study. A number of studies had also failed to show any significant association between Lp(a) and IHD among T2DM subjects without any emphasis on the reasons behind such findings^{5,16}. On the contrary others have reported significant association between Lp(a) and IHD⁶.

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