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ORIGINAL ARTICLE

# Apo Lipoprotein A1 and High Density Lipoprotein As Risk Marker Among Myocardial Infarction Patients in General Hospital in Kerbala City

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**ABSTRACT**

**Objectives:** All regions of the world has ability to cause presumed disease load from ischemic heart disease and can expect fundamental raise in together morbidity and mortality rates , it is expecting that jointly morbidity and mortality rates from ischemic heart disease will multiple through 1990 and 2020. This study was designed to estimate some biochemical marker, Apo lipoprotein A1, high density lipoprotein among patients with Acute myocardial infarction compared to healthy control.

**Methods:** A case control study was conducted in Al-Husain general hospital in Karbala city, it included forty four cases with recent myocardial infarction and similarity number of age and sex matched healthy controls. The healthy controls group were chosen from medical staff and relatives with no sign and symptoms of coronary heart disease.

**Results:** The study shows a highly significant decrease in the level of Apo lipoprotein A1 and high density lipoprotein cholesterol among acute myocardial infarction patients compared with healthy controls P. value(0.0001).

**Conclusion:** The preserved association of ApoA1 with risk of AMI. As well as Low Apo A1 & HDL-cholesterol levels among MI cases compared with controls study was presenting.



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## INTRODUCTION

Epidemiologic studies carried out during the 1950s & 1960s pointed between correlation fat in the diet, cholesterol in the serum of patients myocardial infarction[1]. Barr *et.al*. in 1951 first described the decreased level of Alpha lipoprotein in myocardial infarction survivors versus asymptomatic control subject. Through the last thirty years, cardiovascular disease (CVD) death rates have turn down in different developed countries, whereas they show to have fundamental raised in various developing countries. All regions of the world has ability to cause presumed disease load from ischemic heart disease and can expect fundamental raise in together morbidity and mortality rates, it is expecting that jointly morbidity and mortality rates from ischemic heart disease will multiple through 1990 and 2020. Approximately 82% of these expected raise in mortality rates and 89% of the expected elevate in morbidity rate from ischemic heart disease will be seen in developing countries [2].

Patients at increased risk of developing Myocardial infarction (MI) include those with multiple coronary risk factors. Generally MI results from risk factors for atherosclerosis. Nearly 10% cases of acute myocardial infarction are caused by non-atherosclerotic factors. With application in screening, diagnosis, prognosis, predicting disease recurrence and therapeutic monitoring, biomarkers have a powerful approach to understand the spectrum of cardiovascular disease (CVD)[3]. There is minimal compact over which of the various lipid fractions and Apo lipoproteins top identify the patients as a matter risk, serum high density lipoprotein (HDL) cholesterol and headway of coronary artery disease [4,5]. Apo lipoprotein AI is a main lipoprotein in HDL cholesterol and its serum concentration identify patients with the elevated coronary risk [6,7]. Yet, there is raising index that the gauge of Apo lipoprotein A-I (Apo A-I), the protein component of HDL may insert worthy knowledge in the clinical appreciation of tendency to CHD [8].

## MATERIALS AND METHODS

Our a case control study in Al Husain teaching hospital which was chosen because it is a central hospital in Karbala city. Patients were taken from cardiac care unite (CCU) in the general hospital. About 15 patients were excluded from the study for having one or more of the following criteria.

**Exclusion criteria:**

- Previous History of IHD.
- Patients with cancer, liver, thyroid, renal and autoimmune disease.
- Patients with drugs that increase LDL cholesterol and decrease HDL cholesterol such (progestin and corticosteroids drug ).

**Inclusion criteria:** All patients with early myocardial infarctions within 24 hours with a positive Troponin test that done to confirm AMI by a simple rapid one –step immunoglobulin for qualitative detection 0f Troponin I test.

Forty four cases of AMI and similarity number of control matched by age and sex and comparable BMI. Controls were selected from attendants of the hospital who were free of CHD, (relatives of patients and staffs) were matched for age and sex with cases of MI.

Weight and BMI, were measured with light clothes and bare footed by the same electronic scale, which measures the weight and height .BMI was collected using the equation, BMI = weight (kg)/ height (m2).

**ApoA1 manual leaflet:** Throughout the manual procedure, all components of kit and serums, unless the (TMB) substrate, should be set to room temperature (18-25ºC) before to use, the (TMB), should preferably be utilize cold.

- The numbers of strips required for the standard curve, assay background control, the blanks and samples assembled in the plate frame.
- 100µl /well of each concentration of diluted Apo A1 standard and assay background controls was added. For the samples was added 50µl assay buffer and 50 µl sample per wells, mixed by tapping the plate and incubated at room temperature for two hours.
- The wells were washed by 5 times (200-300µl) with diluted wash buffer.
- 100µl from detection Ab. was added to each wells, covered the plate with adhesive plate cover and incubated at room temperature for 60 min.
- The wells were washed by 5 times (200-300µl) with diluted wash buffer.
- 100µ of (SA-HRP) was added and covered the plate and incubated at room temperature at 60 min.
- The wells were washed by 5 times (200-300µl) with diluted wash buffer.
- 100 µl of TMB was added to all wells and incubated at room temperature in dark for 15min.
- 100µl of (stop solution) was added to all wells and measured at 450nm absorbance in micro plate reader within 15min.

**HDL Procedure:**

1. **Precipitation.**
   - Reagent and samples to room temperature was brought.
   - Pipette in to labeled centrifuge tube

| | |
|---|---|
| Sample or standard | 0.2 ml |
| Precipitation reagent | 0.4 ml |

   - Mixed and allowed to stand for 10 min at room temp.
   - Centrifuge at 10 min at 4000r. p.m.
   - Separate off the clear supernatant with 2 hr.

2. **Colorimetry.**
   - Cholesterol MR monoreagent and standard 50 mg/dl of the kit to room temperature was brought.
   - Pipette in to labeled tube.

| Tubes | Blank | Samples supernatant | Standard supernatant |
|---|---|---|---|
| **Monoreagent** | 1ml | 1ml | 1ml |
| **supernatant** | - | 50µl | - |
| **standard** | - | - | 50µl |

   - Mixed and late tubes stand for 10 min at room temperature or 5 min at 37C.
   - Read the absorbance (A) of supernatant and standard at 550 nm against reagent blank .

Calculated by Supernatant/standard x concentration of standard =mg /dl HDL.

## RESULTS

In Table 1 and Figure 1 showed decrease level of Apo A1 in MI patients mean (101.30±9.81), range of (88-125) mg/dl in comparison with healthy controls mean (162.16±7.43), range of (139-175) mg/dl with highly significant difference P value 0.0001.

Table 1. **APO A1 level in cases & controls.**

| Lipid profile | Cases | | Controls | |
|---|---|---|---|---|
| | No | % | No | % |
| **APO A1 (mg/dl) Risky (<110)** | 34 | 77.3 | - | - |
| **Normal** | 10 | 22.7 | 44 | 100 |
| **Mean ±SD (Range)** | 101.30±9.81 (88-125) | | 162.16±7.43 (139-175) | |
| **P value** | 0.0001* | | | |

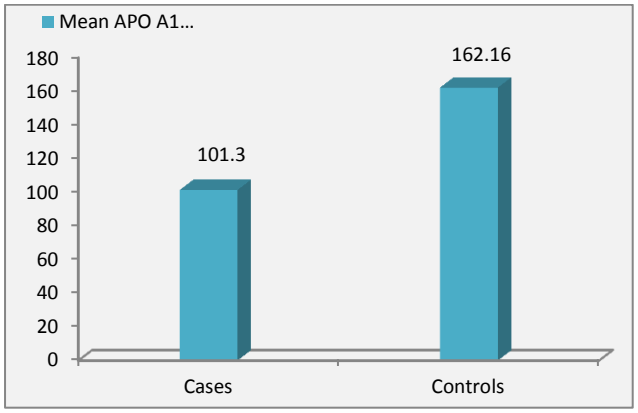


Figure 1. **APO A1 level in cases and controls .**

In the Table 2 and Figure 2 showed significant decrease in the mean of serum HDL in MI cases (29.91±2.74) , range (26-39)mg/dl than in controls (51.45±2.76), range (44-57) P value 0.0001.

Table 2. **Serum HDL level in cases and controls.**

| Lipid profile | Cases | | Controls | |
|---|---|---|---|---|
| | No | % | No | % |
| **HDL (mg/dl) Risky (<35)** | 41 | 93.2 | - | - |
| **Normal** | 3 | 6.8 | 44 | 100 |
| **Mean ± SD (Range)** | 29.91±2.74 (26-39) | | 51.45±2.76 (44-57) | |
| **P value** | 0.0001* | | | |

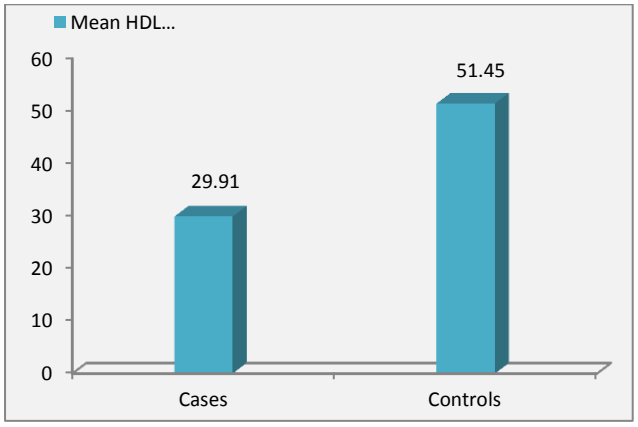


Figure 2. **Serum HDL level in cases and controls**

## DISCUSSION

In our study the level of ApoA1 decreased significantly among AMI patients compared with healthy control was P value 0.0001 with mean level (101.30±9.8) mg/dl and (162.16±7.43) mg/dl respectively . This is in agreement with Abdul Karim [9] study in Iraq in 1989 which reported that the ApoA1 level was significantly lower in the serum of the Myocardial survivors contrasted to healthy persons with P value 0.001. Zaid S. *et.al.* [10] in Kuwait in 1983 showed decrease level of ApoA1 in MI cases vs. controls and it was concluded that the best combination of factors predicting myocardial infarction was ApoA1 and Apo lipoprotein B. Also Sharma and Sniph [11] in India in 2001 found a significant decrease in ApoA1 level among AMI patients compared with controls with P value 0.0001. Schemit *et.al.* [12] in 1985 in US had failed to detect significant difference in ApoA1 level between cases and control due to entrants advertised lipid-decrease level therapy, and women declared menopausal status and use of hormone replacement therapy.

On the other hand my study shows In the Table 2 a highly significant decrease in level of serum high density lipoprotein-cholesterol among acute MI patients vs. healthy controls with P value 0.0001. Whatever that agreement with Ahmed [13] in Iraq in 2008 that showed highly significant decrease in level of HDL-C among acute myocardial patients compared with healthy controls with P value 0.001. In Kuwait N. Al-Mohtaseb *et.al* [14] in 1989 study observed that high density lipoprotein- cholesterol were significantly lower in the young myocardial survivors compared to the controls subject was P value 0.001. Also in Saudi Arabia Mansour [15] in 2004 that showed the mean HDL–C value were statistically lower in subject with coronary artery disease as compared to subject without coronary artery disease with P value 0.024. This study disagreed with Aparna R. *et.al.* [16] in India in 2009 which showed no significant difference in HDL-C level in MI patients compared with controls was P value >0.05 that indicated by Despite low meat and fish consumption, even among non-vegetarians in India, as a cause of it [17].

## CONCLUSIONS

This study reaffirms the importance of hyperlipidemia as risk factors for AMI. The preserved association of ApoA1 with risk of AMI. As well as Low Apo A1 and HDL-cholesterol levels among MI cases compared with controls study was found.

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