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# Admission high density lipoprotein and left main coronary disease in acute coronary syndromes: A prospective cohort from Iraq

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**Abstract**
**Background:** Both low-density lipoproteins and high-density lipoproteins have been proven in epidemiologic research to be significant risk factors for cardiovascular disease.
**Aim of study:** To determine the relation between the level of admission high-density lipoproteins in patients with acute coronary syndrome and coronary artery lesion severity, in-hospital and one-month mortality.
**Methods:** A prospective study that was conducted at IBN-Albitar Center during a period of 11 months from 1st of February 2024 till 1st of January 2025. It included 164 patients diagnosed with acute coronary syndrome who underwent coronary angiography during hospitalization. Electrocardiogram was done for all study patients; Echocardiography was done for all patients within 24 hrs from admission. A blood sample was taken from all patients to perform investigation: Troponin level detection, s. high density lipoprotein and other lipid profile within 24 hrs from admission. The level of s. high density lipoprotein was compared according to the outcome and severity of angiography.
**Results:** In this study, 7.3% of study patients died during hospitalization, while no death reported during one-month of management. No statistically significant differences in means of high-density lipoprotein, low density lipoprotein, total cholesterol, and triglyceride between patients who died and patients who survived during hospitalization. Mean of high-density lipoprotein level was significantly lower in patients with LMS lesion than those without LMS lesion.
**Conclusion:** Lower high-density lipoproteins levels at admission in patients with acute coronary syndrome was significantly related to the presence of LMS disease. Lower high-density lipoproteins level noticed in all in-hospital deaths, however no statistically significant relation could be found.

**Keywords:** Iraq, acute coronary syndrome, high density lipoprotein, angiography, mortality

## Introduction
Death from coronary artery disease (CAD) is still at the top of the global to-do list. Heart failure, unstable angina, Acute Coronary Syndromes (ACS), and unexpected mortality are some of the clinical signs [1]. Death from Coronary Artery Disease (CAD) is still at the top of the global to-do list. Heart failure, unstable angina, Acute Coronary Syndromes (ACS), and unexpected mortality are some of the clinical signs [2]. Without immediate medical attention, over 25% of myocardial infarction instances result in death. Within the first twenty-four hours after symptoms appear, 50% of fatalities occur, and 40% of people afflicted die within the first thirty days. Those who make it to the hospital have a considerably better prognosis, with a 28-day survival rate of over 85%. The mortality rate for patients with unstable angina is about half that of patients with myocardial infarction. Irrespective of the severity of myocardial infarction, arrhythmia is the leading cause of premature death [3]. Proteins called apolipoproteins with lipids like cholesteryl esters, free cholesterol, and phospholipids form a spherical micelle called a high-density lipoprotein (HDL) [4]. There may be more reasonable ways to approach lipid-modifying treatment if we knew more about the associations between plasma lipids or lipoproteins and post-ACS prognosis. There is some evidence that ACS triggers an acute-phase systemic inflammatory response, which can alter lipid and lipoprotein concentrations in circulation [5]. A drop in HDL-C follows an ACS event.

This level starts to drop within the first twenty-four hours following an ACS event, reaches rock bottom around the one-week mark, and then starts to rise again gradually as metabolic steady state is achieved within the one-month mark [6, 7]. Reduced levels of LDL-C are more noticeable than reduced levels of HDL-C. It has been suggested that following ACS, HDL's anti-inflammatory, cholesterol efflux, and antioxidant capabilities are changed [8]. The magnitude of change in lipoprotein concentrations is related to the extent of myocardial necrosis [9]. It is recommended to take precise measurements either immediately following presentation or a few weeks later due to the acute-phase effects on lipids and lipoproteins. It may be safely assumed, however, that metabolic steady state LDL-C levels will nearly surely be greater than optimal if they are during the acute-phase reaction following an ACS episode [10]. The aim of this study is to correlate between the level of admission high-density lipoproteins in patients with acute coronary syndrome and coronary artery lesion severity, in-hospital and one-month mortality.

## Materials and Methods

### Study design, setting and time

From February 1, 2024, to January 1, 2025, a prospective study was implemented by researchers at Iraq's Ibn-Al Bitar Centre for Cardiac Surgery.

### Study Population

The study included 164 patients diagnosed with ACS including STEMI, NSTEMI, and UA who underwent coronary angiography during hospitalization. They were informed about the nature of the study and verbal consent was obtained from them. Diagnosis of ACS was based on electrocardiogram, clinical symptoms and cardiac markers. It can be divided into three categories: STEMI, NSTEMI, and UA. Patients with more than 24 hrs. of presentations and those with a known case of hereditary hypercholesterolemia were excluded from this study. A questionnaire was applied to all enrolled patients including questions to gather the following information: Age and gender, risk factors (Hypertension, diabetes mellitus, smoking, history of IHD, and family history of IHD), use of statin, clinical symptoms (chest pain, shortness of breath, palpitation, syncope, and other symptoms in the cardiovascular system). This questionnaire included measuring weight and height to calculate Body Mass Index (BMI) level. Patients were classified according to BMI as: Normal ($\leq$ 24.99 kg/m$^2$), overweight (25-29.99 kg/m$^2$), and obese ($\geq$ 30 kg/m$^2$).

### Workup

- Electrocardiogram was done for all study patients.
- Echocardiography was done for all patients within 24 hrs. from admission by Echo machine GE vivid E9 to assess the Left Ventricular function by ejection fraction, regional wall motion abnormalities, and any mechanical complications).
- Left Ventricular function was classified according the following (Table 1) [11]:
- Coronary angiography with or without Percutaneous Coronary Intervention was done through the femoral approach by seldinger technique using Philips catheterization machine. All the operations and the decisions were done under direct supervision of the consultant operator.
- A blood sample was taken from all patients to perform the following investigation:
  - Quantitative Troponin test.
  - Lipid profile within 24 hrs from admission.
  - Complete blood count, renal function test, random blood sugar, and viral screen (routinely).
- Results of the coronary angiography were taken according to:

**Type of the vessel lesion**

- Left Main Stem (LMS).
- Left Anterior Descending Artery (LAD).
- Left Circumflex Artery (LCx).
- Right Coronary Artery (RCA).

**Number of vessel lesions**

- Single vessel disease
- Two vessel disease
- Three vessel disease

The outcome of this study included mortality that was followed and recorded directly in-hospital during admission and one month by telephone contact.

### Ethical Considerations

The Declaration of Helsinki lays forth the ground rules for how this study should be carried out ethically. Approval from Ibn Sina University of Medical and Pharmaceutical Sciences Ethics Committee was obtained. After all patients or their surrogates were informed about the objectives and procedures of the study, we acquired a written informed consent from them. Data security was guaranteed by utilizing identifying coding and storing data in a password-protected environment.

### Statistical analysis

Statistical Package for Social Sciences (SPSS) version 26 was used to analyze the data. The information is shown as the average, the standard deviation, and the range. Information is organized into categories with percentages and frequencies displayed. The continuous variables were compared using an independent t-test with two tails. For statistical significance, a P-value of less than 0.05 was used.

## Results

In this study, mean of age was 55.63±8.7 years; 78% were males; 43.9% were overweighed; 48.8% were current smokers; 36.6% were hypertensive; 36.6% were diabetics; and 7.3% were using anti-statin drugs. IHD was presented in 12.2% of study patients and family history of IHD was documented in 9.8% of cases (Table 2).

In the current study, LMS lesion occurred in 7.3% of study patients, while LAD lesion occurred in 78%. LCX lesion was noticed in 39% of study patients, while RCA lesion was noticed in 43.9%. We noticed that 36.6% of study patients had single affected artery; 7.3% had died during hospitalization, while no death reported during one-month of management (Table 3).

No statistically significant differences in means of HDL, LDL, total cholesterol, and TG between patients who died and patients who survived during hospitalization (Table 4).

Mean of HDL level was significantly lower in patients with LMS lesion than those without LMS lesion (33 versus 36.19 mg/dl, P=0.001). No statistically significant differences ($p \geq 0.05$) in mean of HDL in all other comparisons (Table 5).

## Discussion

The lipid content of atherosclerotic plaques is connected to their sensitivity to rupture and initiate ACS, and plasma lipoproteins are implicated in the origin of atherosclerosis [12]. Anti-inflammatory and antioxidant actions are two of HDL's roles that have recently gotten a lot of attention. HDL's anti-oxidant and anti-inflammatory properties have been linked to a reduced risk of cardiovascular disease [13].

In this study, HDL level was low in 72% of study patients; LDL was high in 53%; total cholesterol level was high in 35.6%; and TG level was high in 51.8% which is different from result obtained by Pintó *et al* study in 2010, in which high LDL found in 66.1%, high TG level observed in 19.4% and mean of cholesterol was 4.87±1.19 mmol/L [14].

In this study, mean of HDL level was significantly lower in patients with LMS lesion than those without (33 versus 36.19 mg/dl, P=0.001). No statistically significant differences in means of HDL, LDL, total cholesterol, and TG between patients who died and patients who survived during hospitalization.

Different results seen in Lee CH *et al* study in 2016, in which patients with a higher HDL-C level at admission showed significantly lower rates of 12-month major adverse cardiac events, particularly cardiac and all-cause mortality. These beneficial effects were observed in the rates of in-hospital mortality and 1-month major adverse cardiac events during the early period after acute MI [15]. Furthermore, Thabet NI *et al* study in 2015 found that impact of HDL-C on in-hospital outcomes revealed that low HDL-C was associated with higher all in hospital morbidity and mortality with a different association in each type, as in form of congestive heart failure (CHF) (P=0.01), recurrent ischemic attacks (P=0.01), re-infarction (P=0.29), cardiogenic shock (P=0.013) and Death (P= 0.012) [16].

Possible causes for the discrepancies between the aforementioned research include different sample sizes, unhealthy eating habits, lack of physical activity, urbanization, economic growth, stress, and physical inactivity. In fact, there is no research that takes into consideration the relation between lipid profile and the site of lesion, so we discuss its relation to ACS.

In Carnuta *et al* study in 2017, results obtained showed that plasma cholesterol levels were significantly decreased in stable angina patients compared to normal subjects, probably due to the intensive treatment with statins, but they were significantly higher in ACS compared to stable angina plasma [17].

Multiple anti-atherogenic effects of the HDL particle are thought to be mediated by its role in eliminating cholesterol from macrophages via "macrophage cholesterol efflux [18]. Furthermore, because cholesterol export capacity has a strong inverse connection with cardiovascular risk, it was predicted that boosting capacity would be another therapeutic target [19]. In addition, the vascular effects of HDL in patients with a variety of cardiovascular diseases differ significantly from those of HDL in healthy subjects, leading to the term "HDL dysfunction [20]. HDL possesses a complement of antioxidant enzymes that act to maintain an anti-inflammatory state in the absence of inflammation. These antioxidant enzymes are inactivated in the presence of systemic inflammation, such as in acute coronary syndrome, and HDL accumulates oxidized lipids and proteins, making it proinflammatory [21]. Some another studies have suggested that measuring the quality and novel functions of HDL could provide an improved means of identifying subjects at increased risk for atherosclerosis [19]. HDL's quality and function are appealing targets for new therapeutics. Other recent research have shown that HDL subclasses, particularly HDL3-C, have an inverse relationship with coronary heart disease [22].

## Study Limitations

This study has a number of limitations. First, the statistical power is limited, particularly for mortality analysis, due to the small sample size (N=164) and single-center design, as well as the low event rate (12 in-hospital deaths). Second, limited generalizability as lifestyle and healthcare context may differ from other settings.

**Table 1:** Left Ventricular function assessment according to the gender

| LV EF% | Normal | Mild dysfunction | Moderate dysfunction | Severe dysfunction |
|---|---|---|---|---|
| Male | 52-72 | 41-51 | 30-40 | < 30 |
| Female | 54-74 | 41-53 | 30-40 | <30 |

**Table 2:** Distribution of study patients by certain characteristics

| Variable | No (N= 164) | Percentage (%) |
|---|---|---|
| **Age (Year)** | | |
| < 50 | 36 | 22.0 |
| 50-59 | 64 | 39.0 |
| ≥ 60 | 64 | 39.0 |
| **Gender** | | |
| Male | 128 | 78.0 |
| Female | 36 | 22.0 |
| **BMI level** | | |
| Normal | 36 | 22.0 |
| Overweight | 72 | 43.9 |
| Obese | 56 | 34.1 |
| **Hypertension** | | |

| | | |
|---|---|---|
| Yes | 60 | 36.6 |
| No | 104 | 63.4 |
| **Diabetes Mellitus** | | |
| Yes | 60 | 36.6 |
| No | 104 | 63.4 |
| **Smoking** | | |
| Current smoker | 80 | 48.8 |
| Non-smoker | 84 | 51.2 |
| **Ischemic heart disease** | | |
| Yes | 20 | 12.2 |
| No | 144 | 87.8 |
| **Family history of IHD** | | |
| Yes | 16 | 9.8 |
| No | 148 | 90.2 |
| **Anti-statin use** | | |
| Yes | 12 | 7.3 |
| No | 152 | 92.7 |
| **Clinical symptom** | | |
| STEMI | 88 | 53.7 |
| Non STEMI | 48 | 29.3 |
| UA | 28 | 17.0 |
| **Ejection fraction (%)** | | |
| Normal (52%-74%) | 54 | 32.9 |
| Mildly abnormal (41%-51%) | 86 | 52.4 |
| Moderately abnormal (30%-40%) | 24 | 14.7 |

**Table 3:** Distribution of study patients by angiographic severity and mortality

| Variable | No (N=164) | Percentage (%) |
|---|---|---|
| **LMS Lesion** | | |
| Yes | 12 | 7.3 |
| No | 152 | 92.7 |
| **LAD Lesion** | | |
| Yes | 128 | 78.0 |
| No | 36 | 22.0 |
| **LCX Lesion** | | |
| Yes | 64 | 39.0 |
| No | 100 | 61.0 |
| **RCA Lesion** | | |
| Yes | 92 | 43.9 |
| No | 72 | 56.1 |
| **Number of affected arteries** | | |
| No affected arteries | 16 | 9.8 |
| Single | 60 | 36.6 |
| Two | 40 | 24.4 |
| Three | 48 | 29.3 |
| **In-hospital mortality** | | |
| Died | 12 | 7.3 |
| Survived | 152 | 92.7 |
| **One month mortality** | | |
| Survived | 164 | 100.0 |

**Table 4:** Comparison in HDL and other lipid profile according to in hospital mortality

| Variable | In hospital mortality | | P-Value |
|---|---|---|---|
| | Died, Mean ± SD | Survived, Mean ± SD | |
| HDL (mg/dl) | 35.33±0.98 | 36.01±7.12 | 0.293 |
| LDL (mg/dl) | 108.66±18.4 | 123.6±32.3 | 0.116 |
| Total cholesterol (mg/dl) | 176.0±88.0 | 185.22±49.9 | 0.726 |
| TG (mg/dl) | 155.0±11.2 | 148.3±42.3 | 0.16 |

**Table 5:** Comparison in HDL level according to angiography severity

| Variable | HDL level (mg/dl), Mean ± SD | P-Value |
|---|---|---|
| **LMS Lesion** | | |
| Yes | 33.0±1.7 | 0.001 |
| No | 36.19±7.1 | |
| **LAD Lesion** | | |
| Yes | 34.22±5.34 | 0.233 |
| No | 36.23±7.1 | |
| **LCX Lesion** | | |
| Yes | 35.16±5.7 | 0.084 |
| No | 37.21±8.3 | |
| **RCA Lesion** | | |
| Yes | 36.77±6.3 | 0.172 |
| No | 35.32±7.2 | |
| **Number of affected arteries** | | |
| No affected arteries | 35.0±6.7 | 0.28 |
| One | 34.86±5.0 | |
| Two | 37.4±7.4 | |
| Three | 36.45±8.2 | |

## Conclusion

Lower HDL levels on admission in patients with ACS were significantly related to the presence of LMS disease. Lower HDL level noticed in all In-hospital deaths, however no statistically significant relation could be found.

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## Conflict of Interest

Not available

## Financial Support

Not available

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