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Original article

Triglyceride and High density lipoprotein Cholesterol ratio in Patients with Acute Coronary Syndrome

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Abstract:

- ***Background*:** Ischemic heart disease is the leading cause of death worldwide. This study assessed the prevalence of elevated triglyceride-to-HDL cholesterol ratio among acute coronary syndrome patients versus a control group, highlighting its role in atherogenesis.
- ***Methods:*** A case-control study included 100 ACS inpatients and 100 non-ACS outpatients at Baghdad Teaching Hospital from July to November 2013. Demographic and clinical data were collected, and lipid profiles analyzed.
- ***Result:*** The TG/HDL-C ratio (≥5) was higher in 58% of ACS patients versus 10% of controls. ACS patients had significantly lower HDL-C and higher TG levels. Triglycerides varied by ACS type, being lowest in unstable angina and highest in STEMI and NSTEMI (*P*=0.031).
- ***Conclusions:*** High TG, low HDL-C, and elevated TG/HDL-C ratio are strongly associated with ACS, indicating their value as risk markers for early detection and prevention.
- **Keywords:** Acute coronary syndrome, Hypertriglyceridemia, Atherosclerosis

INTRODUCTION

Ischemic heart disease (IHD) remains one of the leading causes of death in both developed and developing countries (1). According to World Health Organization (WHO) statistics, age-adjusted mortality rates due to IHD are among the highest globally (2), and projections indicate that IHD will become the foremost cause of death in developing nations by 2020 (3,4). This trend is largely attributed to the high prevalence of IHD risk factors (5). In the United States, IHD is the primary cause of mortality in adults over 35 years, accounting for nearly one-third of all deaths in this age group (6).

IHD is characterized by narrowing of the coronary arteries, leading to reduced oxygen and nutrient supply to the heart muscle, ultimately causing myocardial infarction. The disease has two notable features: it often progresses silently until advanced stages, and its clinical manifestations are nonspecific, overlapping with other pathologies. While mortality due to IHD has declined in North America and Western Europe due to improved treatment strategies, it continues to rise in Asia and Eastern Europe (7).

IHD is driven by a combination of genetic and environmental factors. Tobacco use, diabetes mellitus (DM), and hypertension are prominent environmental contributors (8).

Most cases have a multifactorial genetic basis, with several genes interacting with environmental exposures to influence disease onset and severity (9). Biochemical mechanisms involved in IHD include lipid and apolipoprotein metabolism, inflammation, endothelial dysfunction, platelet activity, thrombosis, fibrinolysis, and blood pressure regulation (10).

Lipid abnormalities play a central role in IHD pathogenesis. Low-density lipoprotein cholesterol (LDL-C) promotes atherosclerosis, whereas high-density lipoprotein cholesterol (HDL-C) offers protection by preventing LDL-C oxidation and deposition in arterial walls. Each 1 mg/dL decrease in HDL-C is associated with a 2–3% increase in IHD risk (11). HDL-C is thus termed "good cholesterol," while LDL-C is considered "bad cholesterol." Unfortunately, data on IHD prevalence and cardiovascular risk profiles in developing countries remain limited (12,13).

Kumar et al. (14) reported significantly elevated total cholesterol (TC) and triglyceride (TG) levels, along with lower HDL-C levels, in acute myocardial infarction (AMI) patients. The association between low HDL-C and increased AMI risk is observed in both Asian and non-Asian populations (15). HDL-C concentrations below 40 mg/dL in men and 50 mg/dL in women, along with TG levels ≥150 mg/dL, are considered

independent risk factors for coronary artery disease (16–21). Prospective studies, such as the Framingham Heart Study and the PROCAM Study, have consistently shown that low HDL-C is independently associated with increased cardiovascular risk (22).

Among diabetic patients, dyslipidemia typically presents as elevated TG—mainly in very-low-density lipoprotein (VLDL)—and reduced HDL-C (23–26). Randomized controlled trials have shown that lipid-lowering therapies improve both mortality and morbidity in individuals with or at risk for IHD (29–31). However, despite achieving target lipid levels, many patients continue to experience major adverse cardiac events (MACE), with reported rates ranging from 8–22% (29–37). Effective management of post-myocardial infarction dyslipidemia remains a critical component of care (38).

The role of lipid fractions—particularly TG and HDL-C—as independent predictors of IHD has gained increasing attention. A lipid profile marked by elevated TG and reduced HDL-C, commonly seen in metabolic syndrome, is now recognized as a potent contributor to IHD, potentially rivaling LDL-C in importance (39).

IHD, a major subtype of cardiovascular disease (CVD), is insidious and often progresses without symptoms, making it a leading cause of morbidity and mortality worldwide (40). Over the past four decades, there has been a growing emphasis on evaluating risk factors in asymptomatic individuals (41). IHD development is characterized by atherosclerotic plaque formation in coronary arteries, originating from lipid accumulation and leading to vascular occlusion, ischemia, or sudden cardiac death. These plaques may rupture, forming clots that obstruct blood flow to the myocardium. Common symptoms include chest pain, nausea, syncope, and anxiety (42).

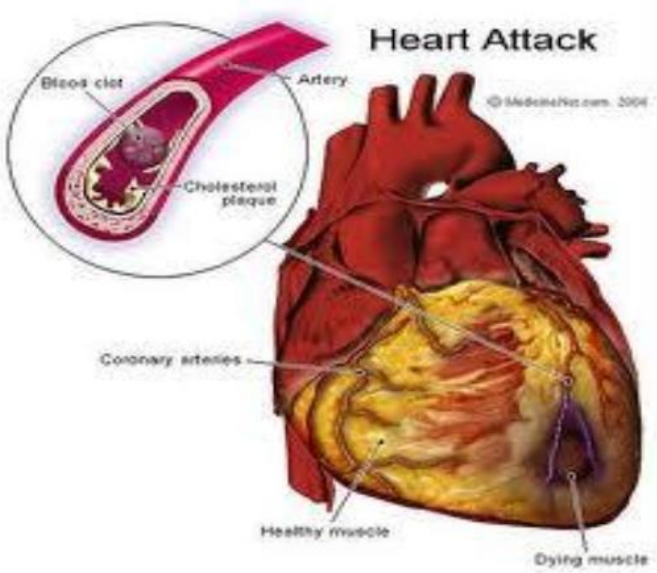

Fig. 1 Coronary arteries and veins (43)

Atherosclerosis, derived from the Greek language, refers to the localized and progressive accumulation of lipids and fibrous tissue within the arterial wall, forming plaques. In the context of coronary arteries, atherosclerosis involves structural changes within the arterial walls that impair or obstruct normal blood flow, leading to myocardial ischemia (44).

This process typically begins with the deposition of cholesterol, lipids, calcium, fibrous connective tissue (such as collagen), and cellular waste products within the arterial layers. Over time, these components accumulate to form fatty plaques, a condition known as atherosclerosis (45) (Fig. 2).

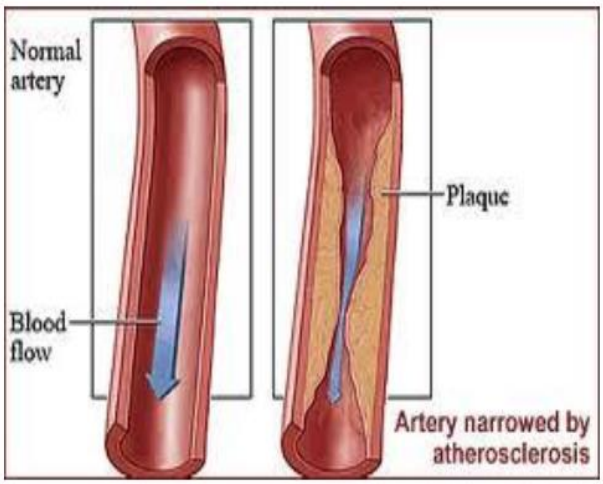

Fig. 2 The difference between normal and narrowing artery (45)

Atherosclerosis develops through a multifactorial interplay of genetic and environmental factors that contribute significantly to its pathophysiology. Environmental influences such as cigarette smoking, hypertension, and high dietary cholesterol intake are strongly linked to an increased risk of ischemic heart disease (IHD). Among these, lipid profile components—including cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C)—have shown consistent

associations with atherosclerosis in numerous epidemiological and genetic studies. Elevated HDL-C levels are known to exert a protective effect against atherogenesis, the process by which lipids accumulate within vessel walls, ultimately leading to arterial narrowing and IHD (46).

Atherogenesis refers to the gradual formation of atherosclerotic plaques within the arterial walls, a chronic condition that typically begins in early life and progresses silently over decades (10).

Modifiable risk factors for IHD include hypertension, hypercholesterolemia, smoking, diabetes mellitus, and elevated body mass index (BMI). Hypertension—defined as systolic blood pressure ≥140 mmHg or diastolic ≥90 mmHg—is a major contributor to vascular damage and IHD, particularly in older adults (47,48). Hypercholesterolemia, caused by genetic or lifestyle factors, is a critical determinant of atherosclerotic plaque formation and is closely associated with increased oxidative stress and LDL-C oxidation (49,50). Familial hypercholesterolemia (FH) represents a primary genetic contributor to premature atherosclerosis (8).

Cigarette smoking is another key risk factor for IHD. With over 4,000 harmful chemicals, tobacco smoke promotes endothelial dysfunction and atherogenesis through nicotine-induced vasoconstriction and carbon monoxide–mediated vascular injury. Smoking alters lipid metabolism, increasing serum cholesterol, triglycerides, and LDL-C, while reducing HDL-C levels. It is estimated that smoking increases the risk of atherosclerosis by 50% and doubles the incidence of IHD (51–53).

Diabetes mellitus (DM), characterized by persistent hyperglycemia, is strongly associated with IHD development. Diabetics face a 3–5 times higher risk of coronary atherosclerosis than non-diabetics, even when controlling for other risk factors (54,55).

Body mass index (BMI), a measure of weight relative to height, is widely used to assess obesity as shown in (Figure 3), which is also a known risk factor for IHD. Overweight is defined as a BMI of 25–30 kg/m² and obesity as ≥30 kg/m². Elevated BMI is strongly linked to metabolic disturbances and increased cardiovascular risk (56,57).

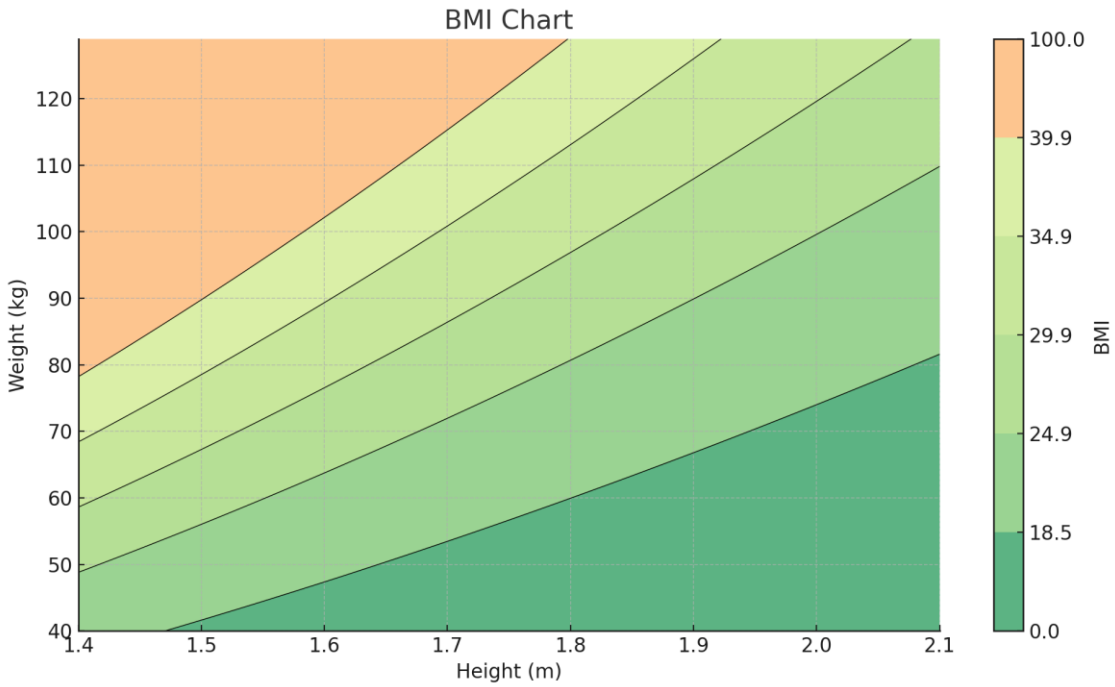

Fig. 3 BMI chart (57)

Non-modifiable risk factors for IHD include age, family history, and sex. Advancing age is a major determinant of cardiovascular risk, with men between 60–64 years showing a sevenfold higher risk compared to those aged 30–34. By age 70, the cumulative IHD risk is 35% for men and 24% for women, rising with age (58). Family history of early-onset IHD suggests a strong genetic predisposition, particularly in younger individuals (8,9). Gender also influences IHD risk; men are generally more susceptible, but women experience increased risk after menopause due to the decline in estrogen's cardioprotective effects (59,60). Women also tend to have higher HDL-C levels than age-matched men, which may confer some protection against IHD (8).

Dyslipidemia—characterized by elevated total cholesterol, triglycerides, and LDL-C, along with low HDL-C—is a central risk factor for IHD. Numerous studies have identified low HDL-C as an independent predictor of cardiovascular events (61,62). The Framingham Heart Study showed that 43–44% of coronary events occurred in individuals with HDL-C <40 mg/dL. Risk is nearly doubled with triglycerides >200 mg/dL or HDL-C <40 mg/dL, and quadrupled when both abnormalities coexist (61). The prevalence of familial hypertriglyceridemia among patients with premature IHD further highlights its importance (48).

Recent observations from Jordan reported that patients with chronic IHD had significantly higher triglyceride and cholesterol levels and lower HDL-C levels compared to non-IHD individuals. While only one-fifth had hypercholesterolemia, half had elevated triglycerides (61).

Lipids and other fats circulate in the blood as lipoproteins, which transport cholesterol and triglycerides in spherical particles composed of a hydrophobic core and a surface layer of apolipoproteins. These include atherogenic particles like apoB (B-48 and B-100), and protective ones like apoA and apoE. Lipoproteins are categorized by density and electrophoretic mobility: VLDL, LDL, IDL, HDL, and lipoprotein(a). Among these,

HDL exerts protective effects against IHD through various mechanisms, while the others contribute to disease progression (63). (Figure 4)

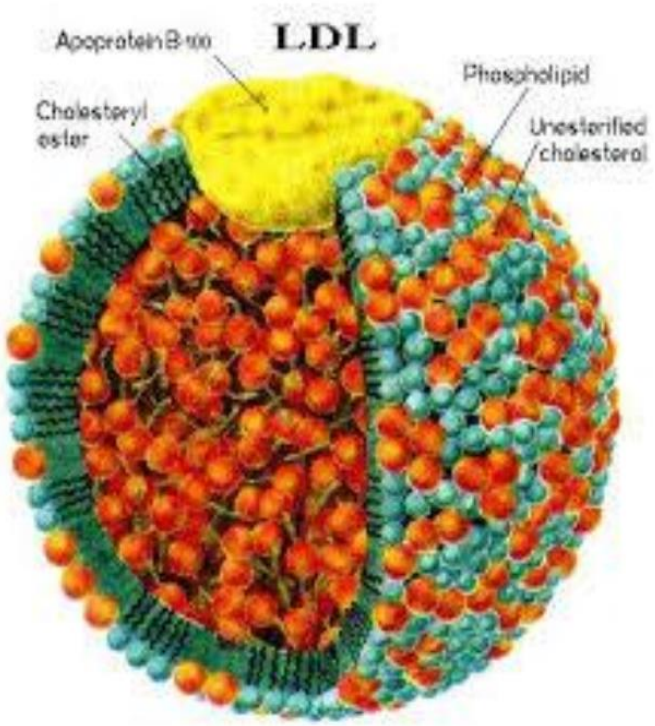

Fig. 4 Low density lipoprotein Composition (64)

Cholesterol in the human body is sourced both externally through dietary intake and internally through synthesis in hepatocytes. As a water-insoluble compound, cholesterol requires transport via lipoproteins. It is present in every cell membrane and plays a vital role in maintaining cell integrity; without it, cells would not retain their spherical structure. Moreover, cholesterol is a precursor for the synthesis of several important hormones and is essential in the formation of key lipoproteins including high-density

lipoprotein (HDL), low-density lipoprotein (LDL), intermediate-density lipoprotein (IDL), and very-low-density lipoprotein (VLDL) (63).

LDL and HDL are particularly significant in the pathogenesis and prevention of atherosclerosis. Oxidized LDL (oxLDL) exerts harmful biological effects on the vascular endothelium by promoting cytokine production, impairing vasodilator function, and stimulating growth factor release. These actions contribute to the onset of atherogenesis by altering the oxidative state within vessel walls, thus LDL-C is often labeled as "bad cholesterol" (50). In contrast, HDL-C serves a protective function, removing excess cholesterol from arterial walls and transporting it to the liver for excretion through metabolic pathways. Due to its anti-atherogenic properties, HDL-C is referred to as "good cholesterol" (48). Findings from the Framingham Heart Study indicate that HDL-C levels ≥60 mg/dL offer significant protection against ischemic heart disease (IHD), even when LDL-C levels are elevated (46). Furthermore, both low HDL-C and high triglyceride (TG) levels have been recognized as independent risk factors for IHD (61).

Triglycerides, which are part of the chylomicron family, contribute to the composition of both LDL and HDL. While the relationship between TG levels and IHD remains complex, evidence increasingly supports the role of elevated TG as an independent risk

factor, especially for premature IHD. European studies, particularly the Prospective Cardiovascular Münster (PROCAM) study, have shown that IHD risk rises proportionally with TG levels up to 800 mg/dL. A TG concentration exceeding 200 mg/dL, especially when combined with low HDL or a high LDL/HDL ratio, significantly increases cardiovascular risk (48).

This study aimed to assess the prevalence of the triglyceride to high-density lipoprotein cholesterol (TG/HDL-C) ratio among 100 patients diagnosed with acute coronary syndrome compared to 100 individuals in a control group.

PATIENT and METHOD

This cross-sectional study was conducted at the Coronary Care Unit (CCU) of Baghdad Teaching Hospital between July and November 2013. A total of 100 patients diagnosed with acute coronary syndrome (ACS) were included. Eligibility criteria required that lipid profiles be obtained within the first 24 hours of hospital admission following a 12-hour overnight fast. Patients whose lipid measurements were taken after this window were excluded, as lipid levels beyond 24 hours from ACS onset may not reflect baseline metabolic status (21–26). Additional exclusion criteria included hypothyroidism, renal disorders such as nephrotic syndrome, liver diseases including hepatitis, chronic alcohol use, and current use of lipid-lowering agents or corticosteroids.

The ACS group was further categorized into subtypes: ST-elevation myocardial infarction (STEMI, n=33), non-ST-elevation myocardial infarction (NSTEMI, n=31), and unstable angina (UA, n=36). A comparison group of 100 individuals without ACS was also included as controls. UA was defined as ischemic chest pain occurring at rest (lasting more than 10 minutes), being of new onset, or showing a crescendo pattern. NSTEMI was diagnosed based on typical symptoms of unstable angina accompanied by elevated cardiac biomarkers. STEMI was confirmed by persistent chest pain lasting

≥30 minutes, ST-segment elevation on ECG (≥0.2 mV in two contiguous precordial leads or ≥0.1 mV in two contiguous limb leads), or new left bundle branch block, along with elevated cardiac troponin I.

Anthropometric measurements included weight and height (with participants wearing light clothing), and blood pressure measured bilaterally using a mercury sphygmomanometer while the patient was in a supine, relaxed state. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Waist circumference was measured at the midpoint between the lower costal margin and iliac crest at the end of exhalation, with patients either standing or lying supine depending on their condition.

Laboratory assessments were conducted using fasting venous blood samples collected in plain vacutainer tubes. Triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) levels were determined enzymatically using kits provided by Human Laboratories (Germany) on an Autoanalyzer system (Roche, Germany). According to ATP III criteria, TG levels ≥150 mg/dL were considered elevated, and HDL-C levels <40 mg/dL for men or <50 mg/dL for women were considered low.

Data were analyzed using SPSS software (version 15 for Windows). Descriptive statistics, including frequencies, percentages, means, and standard deviations (SD), were computed. Categorical variables between the two groups were compared using the Chi-square test. For continuous variables, independent two-sample t-tests were applied to assess differences between ACS patients and controls, while one-way ANOVA was used to compare variables among the three ACS subgroups. A p-value ≤ 0.05 was considered statistically significant, and $p \leq 0.01$ was deemed highly significant. The results were summarized and illustrated using tables and figures, each accompanied by descriptive explanations.

RESULTS

A total of 100 patients with acute coronary syndrome (ACS) and 100 control individuals were enrolled in the study. The demographic and clinical characteristics of the study groups are summarized in Table 1.

1. **Age and Sex Distribution**

   The patient group included 63 males and 37 females, while the control group comprised 64 males and 36 females. The mean age was 60.7 ± 8.2 years in the patient group and 58.3 ± 9.3 years in the control group. There were no statistically significant differences between the two groups regarding age or sex distribution ($P > 0.05$ for both comparisons).

2. **Waist Circumference and Body Mass Index (BMI)**

   In male participants, the mean waist circumference was 96.5 ± 12.7 cm in the patient group and 93.5 ± 8.1 cm in the control group. Among females, it was 93.3 ± 11.9 cm in patients and 86.6 ± 6.4 cm in controls. Although waist circumference was slightly higher in the patient group, the differences were not statistically significant in either sex ($P > 0.05$). Similarly, BMI values did not show significant differences between the two groups ($P > 0.05$).

3. Smoking Status

Among patients, 57 individuals were smokers, compared to 46 smokers in the control group. This difference was not statistically significant (P > 0.05).

Table 1. Demographic characteristics of study groups

Variable	Cases		Control		P
	No.	%	No.	%	
Sex					
Male	63	63.0	64	64.0	0.88
Female	37	37.0	36	36.0	
Age (years)					
40–49	13	13.0	19	19.0	0.65
50–59	36	36.0	31	31.0	
60–69	34	34.0	35	35.0	
≥ 70	17	17.0	15	15.0	
Mean ± SD	60.7 ± 8.2		58.3 ± 9.3		0.52
Range	46 – 75		41 – 73		
Mean waist circumference (cm)					
Male	96.5 ± 12.7		93.5 ± 8.1		0.27
Female	93.3 ± 11.9		86.6 ± 6.4		0.12
BMI (kg/m²)					
< 25	20	20.0	24	24.0	0.53
25 – 29.9	46	46.0	49	49.0	
≥ 30	34	34.0	27	27.0	
Mean ± SD	27.4 ± 2.8		26.7 ± 4.7		0.38
Smoking					
Yes	57	57.0	46	46.0	0.12
No	43	43.0	54	54.0	

4. History of Chronic Diseases:

A positive history of hypertension, diabetes mellitus (DM), and ischemic heart disease (IHD) was reported in 47, 36, and 32 patients, respectively, compared to 35, 28, and 21 individuals in the control group, as presented in Table 2. There were no statistically significant differences between the patient and control groups in the frequency of these chronic conditions, with all comparisons showing $P > 0.05$.

Table 2. Frequency Distribution of History of Chronic Diseases Among Studied Groups

History		Cases		Control		Total		P
		N	%	N	%	N	%	
Hypertension	Positive	47	47.0	35	35.0	82	41.0	0.14
	Negative	53	53.0	65	65.0	118	59.0	
Diabetes	Positive	36	36.0	28	28.0	64	32.0	0.29
	Negative	64	64.0	72	72.0	136	68.0	
IHD	Positive	32	32.0	21	21.0	53	26.5	0.11
	Negative	68	68.0	79	79.0	147	73.5	

5. Types of Acute Coronary Syndrome (ACS):

The distribution of ACS subtypes among the patients is illustrated in Figure 5. Of the total cases, 36 were diagnosed with unstable angina (UA), 33 with ST-elevation myocardial infarction (STEMI), and 31 with non-ST-elevation myocardial infarction (NSTEMI).

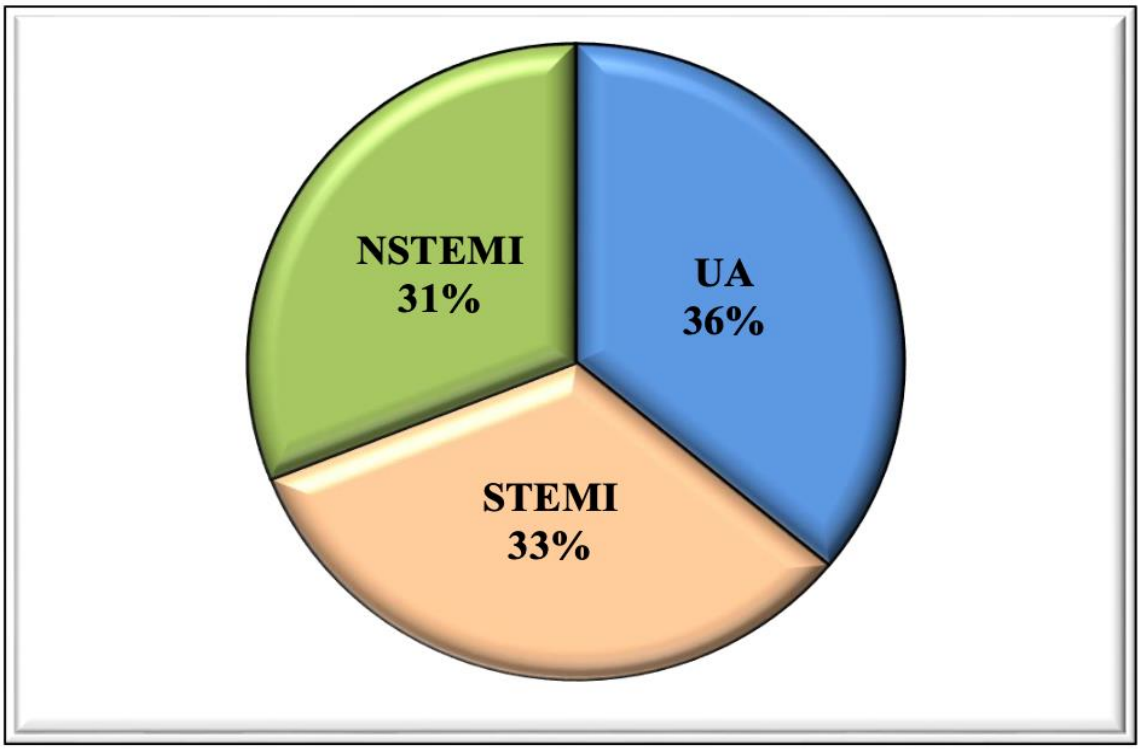

Figure 5. Distribution of Types of acute coronary syndrome in 100 patients with acute coronary syndrome.

6. Comparison of Mean HDL-C and Triglyceride Levels Between Study Groups:

As shown in Table 3, the mean HDL-C levels were significantly lower in both male and female ACS patients compared to their respective controls. In male patients, the mean HDL-C was 37.5 ± 9.4 mg/dL versus 48.6 ± 7.3 mg/dL in male controls ($P < 0.001$), while in female patients it was 37.1 ± 7.8 mg/dL compared to 51.1 ± 9.3 mg/dL in female controls ($P < 0.001$). Additionally, the mean triglyceride level was significantly higher in the patient group (207.8 ± 64.7 mg/dL) compared to the control group (131.9 ± 39.1 mg/dL), also with $P < 0.001$.

Table 3. Comparison of Mean High-Density Lipoprotein Cholesterol (According to Sex) and Triglyceride Between Study Groups

Variable		**Cases**	**Control**	**P**
		Mean ± SD	**Mean ± SD**	
HDL (mg/dl)	Male	37.5 ± 9.4	48.6 ± 7.3	**<0.001***
	Female	37.1 ± 7.8	51.1 ± 9.3	**<0.001 ***
Triglycerides (mg/dl)		207.8 ± 64.7	131.9 ± 39.1	**<0.001 ***

**hs = highly significant.*

7. Distribution of HDL-C Categories in Patients and Controls by Gender:

As illustrated in Figure 6, there was a highly significant difference in HDL-C distribution between patients and controls. Among ACS patients, 42% of males had HDL-C levels < 40 mg/dL and 36% of females had levels < 50 mg/dL, compared to only 8% and 16%, respectively, in the control group. Conversely, the proportion of individuals with HDL-C ≥ 40 mg/dL in males and ≥ 50 mg/dL in females was higher in the control group (56% and 20%, respectively) than in the patient group (21% and 1%, respectively).

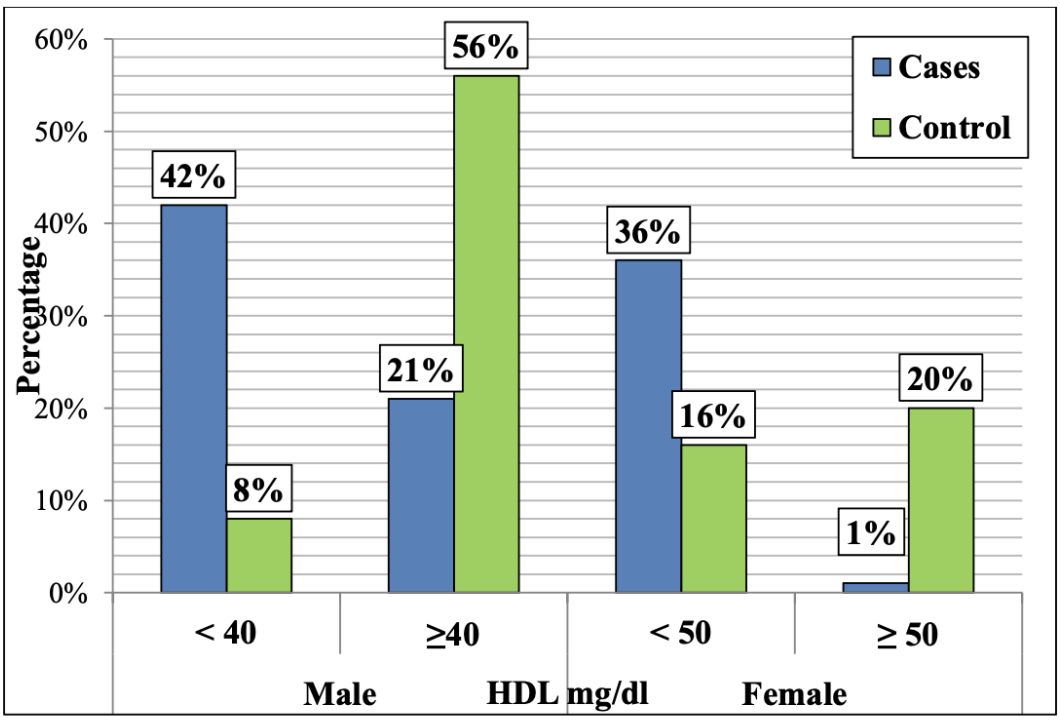

Figure 6. Distribution of high density lipoprotein cholesterol categories in patients and control according to gender.

8. Distribution of Triglyceride Categories Among Study Groups:

As shown in Figure 7, there was a highly significant difference in triglyceride levels between the study groups. Among ACS patients, 72% of males and 30% of females had triglyceride levels ≥ 150 mg/dL, compared to 28% and 8% in the control group, respectively. Conversely, the proportion of individuals with triglyceride levels < 150 mg/dL was higher in the control group (70% of males and 28% of females) than in the patient group (28% and 8%, respectively).

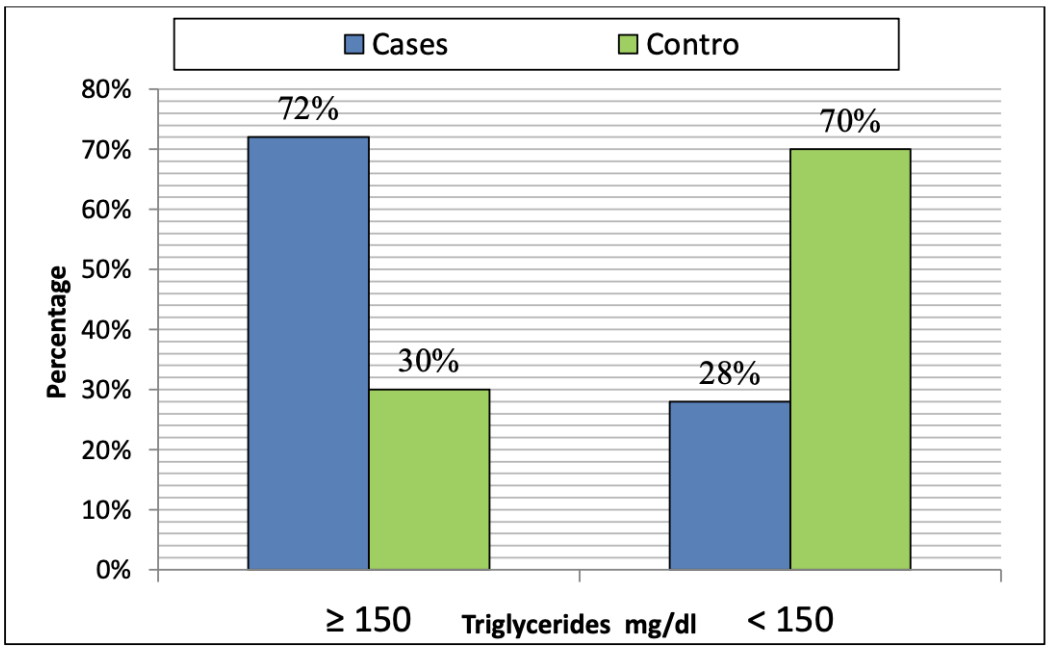

Figure 7. Distribution of triglyceride categories among study groups.

9. Comparison of Mean HDL-C and Triglyceride Levels Among ACS Patients by ACS Type:

Analysis of variance (ANOVA) was used to compare mean HDL-C and triglyceride (TG) levels among ACS patients with different ACS subtypes. No statistically significant differences were observed in mean HDL-C levels across the ACS types in either sex ($P > 0.05$), as shown in Table 4. However, a significant difference in triglyceride levels was found among the ACS subtypes ($P = 0.031$). Patients with unstable angina had lower mean TG levels (187.6 ± 62.0 mg/dL) compared to those with STEMI (218.8 ± 63.3 mg/dL) and NSTEMI (219.5 ± 62.3 mg/dL).

Table 4. Comparison of Mean HDL-C (According to Sex) and Triglyceride According to Types of ACS

Parameter		**UA**	**STEMI**	**NSTEMI**	**P**
HDL-C	Male	37.9 ± 9.4	37.7 ± 10.4	36.5 ± 8.0	0.91
	Female	39.1 ± 7.1	36.3 ± 8.7	36.0 ± 8.7	0.60
Triglycerides		187.6 ± 62	218.8 ± 63.3	219.5 ± 62.3	**0.031**

10. Comparison of TG/HDL-C Ratio Between Study Groups:

A significant difference was observed in the TG/HDL-C ratio between ACS patients and controls. The mean ratio in ACS patients was 6.1 ± 2.3, compared to 2.8 ± 1.2 in the control group. Additionally, 42 ACS patients (42%) had a TG/HDL-C ratio < 5, whereas 90 individuals (90%) in the control group fell into this category. Conversely, a ratio ≥ 5 was found in 58 patients (58%) and only 10 controls (10%). This difference was statistically significant ($P = 0.001$), as shown in Table 5.

Table 5. Comparison of Triglyceride/High-Density Lipoprotein Cholesterol Ratio Between Studied Groups

TG/HDL-C ratio	Cases		Control		Total		P.value
	No.	%	No.	%	No.	%	
< 5	42	42.0	90	90.0	132	66.0	0.001
≥ 5	58	58.0	10	10.0	68	34.0	
Total	100	100.0	100	100.0	200	100.0	
Mean ± SD	6.1 ± 2.3		2.8 ± 1.2		4.5 ± 1.8		0.001

DISCUSSION

In this study, serum lipid profile classifications were based on international guidelines, notably the National Cholesterol Education Program (NCEP) Adult Treatment Panel III, which defines optimal triglyceride (TG) levels as <150 mg/dL, borderline high as 150–199 mg/dL, and high as >199 mg/dL. For HDL-C, optimal levels are ≥40 mg/dL for males and ≥50 mg/dL for females. These same cutoffs have been adopted in regional studies conducted in Jordan (60), Tehran (6), Qazvin (7), Jerusalem and Israel (65), Isfahan (67), Saudi Arabia (69,70), and Gaza (73).

In our study, the male-to-female ratio among ACS patients was 63% to 37%, compared to 64% to 36% among controls, with no statistically significant difference (P>0.05). This contrasts with the Qazvin study, which reported nearly equal distribution between sexes (50.2% vs. 49.8%) (7), the Tehran study (41% male vs. 59% female) (6), and the Jordanian study, where males represented 78.3% (60).

The mean age of ACS patients in our study was 60.7 ± 8.2 years, comparable to the Gaza study (57.27 ± 12.78 years) but higher than in the Tehran study (54 ± 12 years) (6). Control participants had a mean age of 58.3 ± 9.3 years. No significant age differences were observed between patients and controls (P>0.05). Most ACS patients

(70%) fell within the 50–69-year age group, consistent with regional trends. However, comparisons with other studies were limited due to varying age group classifications (6,7,60,65).

Regarding BMI, the average in ACS patients was 27.4 ± 2.8 kg/m², aligning with findings from the Gaza (28.7 kg/m²), Tehran (28.2 ± 4.6 kg/m²) (6), Qazvin (27.5 kg/m²) (7), and Jordan studies (27.5 kg/m²) (60). There was no significant BMI difference between patients and controls (P>0.05). Similarly, waist circumference was higher among ACS patients in both sexes but did not reach statistical significance.

Smoking prevalence among ACS patients was 57%, higher than in Jordan (28%) (60), Qazvin (16.1%) (7), and Palestinian and Jewish populations (21% and 26%, respectively) (65), but lower than in Tehran (88%) (6). Although a higher smoking rate was observed in the ACS group, the difference was not statistically significant (P=0.12). Current smoking was defined as regular smoking within the previous six months, consistent with international definitions (51).

Hypertension was present in 47% of ACS patients, exceeding the prevalence in Tehran (38%) (6), though slightly lower than the non-hypertensive ACS subgroup. Diabetes mellitus was reported in 36% of patients, lower than in Jordan (51%) (60) but higher than in Qazvin (13%) (7). A history of IHD was found in 32% of ACS patients and 21% of controls, indicating a higher prevalence among those with recurrent disease.

ACS subtypes in the study included 36 cases of unstable angina (UA), 33 of ST-elevation myocardial infarction (STEMI), and 31 of non-ST-elevation myocardial infarction (NSTEMI). The overall prevalence of hypertriglyceridemia (TG ≥150 mg/dL) was 72%, surpassing findings from Qazvin (53.5%) (7) and Jordan (55%) (60). The mean TG level was significantly higher in ACS patients (207.8 ± 64.7 mg/dL) than in controls (131.9 ± 39.1 mg/dL, $P<0.001$), comparable to the Jordanian mean of 176 mg/dL (60), but higher than the Gaza figure of 146 mg/dL (73). These findings support previous work suggesting that elevated TG is a meaningful predictor of IHD risk (66), though some literature disputes its independent predictive power (48).

Triglyceride levels also varied significantly across ACS subtypes: UA patients had the lowest mean TG (187.6 ± 62 mg/dL), while STEMI and NSTEMI patients had higher values (218.8 ± 63.3 mg/dL and 219.5 ± 62.3 mg/dL, respectively).

Low HDL-C levels (<40 mg/dL in men, <50 mg/dL in women) were observed in 46% of male and 36% of female ACS patients. These figures are lower than those in Qazvin (62% and 47%) (7), Tehran (6), and Iran (67), but align closely with Jordanian male values (54%) and exceed female values (27%) (60). In contrast, Saudi Arabia reported lower rates in both sexes (28%) (69,70), and U.S. estimates were significantly lower (18% in men, 6% in women) (71). The combination of low HDL-C and elevated TG is increasingly recognized as a significant risk pattern for atherosclerosis, particularly in Middle Eastern populations (60).

Mean HDL-C levels among ACS patients were 37.5 ± 9.4 mg/dL for men and 37.1 ± 7.8 mg/dL for women, closely aligning with Iranian figures (34.5 mg/dL for men, 39.0 mg/dL for women) (67), but lower than those reported in Mediterranean Europe (Italy: 46.2 mg/dL; Spain: 46.6 mg/dL; France: 51.1 mg/dL) (68), and in Jordan (39.0 mg/dL for men, 46.5 mg/dL for women) (60).

No significant differences in HDL-C levels were found between ACS subtypes. In UA patients, mean HDL-C was 37.9 ± 9.4 mg/dL in men and 39.1 ± 7.1 mg/dL in women; in STEMI patients, 37.7 ± 10.4 mg/dL (men) and 36.3 ± 8.7 mg/dL (women); in NSTEMI patients, 36.5 ± 8.0 mg/dL (men) and 36.0 ± 8.7 mg/dL (women).

The mean triglyceride-to-HDL-C ratio was 6.1 ± 2.3, higher than that reported in South India (5.4) (72) and Tehran (4.7) (6). The ratio was ≥5 in 58% of ACS patients compared to only 10% in controls, highlighting its potential value as a predictor for ischemic heart disease.

These findings suggest that hypertriglyceridemia, low HDL-C, and elevated TG/HDL-C ratios are significant contributors to ACS in the studied population. Given the regional variations and growing burden of cardiovascular disease, particularly in Middle Eastern countries, these lipid parameters warrant greater attention in both screening and prevention strategies.

CONCLUSION

This study demonstrates that hypertriglyceridemia is significantly associated with an increased risk of developing acute coronary syndrome (ACS). Additionally, reduced serum levels of high-density lipoprotein cholesterol (HDL-C) were also found to be independently linked with ACS occurrence. The triglyceride to HDL-C ratio emerged as another important indicator, showing a strong association with the development of ACS. Furthermore, a significant variation in triglyceride levels was observed among different ACS subtypes, with patients diagnosed with unstable angina exhibiting lower triglyceride levels compared to those with ST-elevated myocardial infarction (STEMI) and non-ST-elevated myocardial infarction (NSTEMI). These findings emphasize the clinical importance of evaluating both individual lipid parameters and their ratios in the early identification and risk stratification of patients susceptible to acute coronary events.

RECOMMENDATIONS

Based on the findings of this study, several recommendations can be made to enhance the understanding and management of acute coronary syndrome (ACS) in the local population. First, additional research involving both ACS patients and healthy individuals is recommended to establish baseline values and better characterize the distribution of risk factors within the population. This will aid in improving diagnostic accuracy, guiding treatment strategies, and enhancing long-term monitoring. Second, specific attention should be given to targeting triglyceride and high-density lipoprotein cholesterol (HDL-C) levels in both primary and secondary prevention programs for coronary artery disease. Finally, further basic science and epidemiological investigations are essential to uncover the underlying mechanisms linking elevated triglycerides and reduced HDL-C to the pathogenesis of ACS, thereby informing future therapeutic interventions and public health strategies.

Ethical Clearance:

- Institutional approval was obtained from the administration of Baghdad Teaching Hospital.
- Verbal informed consent was obtained from all patients.
- Patients were managed ethically and respectfully by the investigator and supervisor.

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Conflicts of interest:

There are no conflicts of interest.

References

1. Gupta R, Gupta VP. Meta-analysis of coronary heart disease prevalence in India. Indian Heart J. 1996;48:241–5.

2. World Health Organization. World Health Statistics 2008 [Internet]. Available from: http://www.who.int/whosis/whostat/EN_WHS08_TOCintro.pdf

3. World Health Organization. The World Health Report 1999: Making a difference – Emerging epidemics and persistent problems. Geneva: WHO; 1999.

4. Murray CJL, Lopez AD, editors. The global burden of disease: A comprehensive assessment of mortality and disability from disease, injuries, and risk factors in 1990 and projected to 2020. Cambridge: Harvard School of Public Health; 1996.

5. Ibrahim MM, Appel LJ, Rizk HH, Helmy S, Mosley J, Ashour Z, et al. Cardiovascular risk factors in normotensive and hypertensive Egyptians. J Hypertens. 2001;19:1933–40.

6. Hadaegh F, Harati H, Ghanbarian A, Azizi F. Prevalence of coronary heart disease among Tehran adults: Tehran Lipid and Glucose Study. East Mediterr Health J. 2009;15(1):157–66.

7. Fakhrzadeh H, Bandarian F, Adibi H, Samavat T, Malekafzali H, et al. Coronary heart disease and associated risk factors in Qazvin: A population-based study. East Mediterr Health J. 2008;14(1):33–42.

8. Stocker R, Keaney JF Jr. Role of oxidative modifications in atherosclerosis. Physiol Rev. 2004;84:1381–478.

9. Freitas A, Mendonça I, Brión M, Sequeira M, Reis R, et al. RAS gene polymorphisms, classical risk factors, and coronary artery disease in the Portuguese population. BMC Cardiovasc Disord. 2008;8:15.

10. Maren S. Genetic evaluation for coronary artery disease. Genet Med. 2003;5(4):269–85.

11. Chapman MJ, Assmann G, Fruchart JC, Shepherd J, Sirtori C. Raising HDL cholesterol with reduction of cardiovascular risk: The role of nicotinic acid. Curr Med Res Opin. 2004;20(8):1253–68.

12. Beaglehole R. International trends in coronary heart disease mortality and incidence rates. J Cardiovasc Risk. 1999;6:63–8.

13. Okrainec K, Banerjee DK, Eisenberg MJ. Coronary artery disease in the developing world. Am Heart J. 2004;148:7–15.

14. Kumar A, Nagtilak S, Sivakanesan R, Gunasekera S. Cardiovascular risk factors in elderly normolipidemic acute myocardial infarct patients: A case-control study from India. Southeast Asian J Trop Med Public Health. 2009;40:581–92.

15. Karthikeyan G, Teo KK, Islam S, et al. Lipid profile, plasma apolipoproteins, and risk of a first myocardial infarction among Asians: An analysis from the INTERHEART study. J Am Coll Cardiol. 2009;53:244–53.

16. Tokuda Y. Risk factors for acute myocardial infarction among Okinawans. J Nutr Health Aging. 2005;9:272–6.

17. Eberly LE, Stamler J, Neaton JD. Relation of triglyceride levels, fasting and nonfasting, to fatal and nonfatal coronary heart disease. Arch Intern Med. 2003;163:1077–83.

18. Hokanson JE, Austin MA. Plasma triglyceride level is a risk factor for cardiovascular disease independent of HDL cholesterol level: A meta-analysis of population-based prospective studies. J Cardiovasc Risk. 1996;3:213–9.

19. Wong ND, Wilson PW, Kannel WB. Serum cholesterol as a prognostic factor after myocardial infarction: The Framingham Study. Ann Intern Med. 1991;115:687–93.

20. Kannel WB, Vasan RS. Triglycerides as vascular risk factors: New epidemiologic insights. Curr Opin Cardiol. 2009;24:345–50.

21. Miller M, Seidler A, Moalemi A, Pearson TA. Normal triglyceride levels and coronary artery disease events: The Baltimore Coronary Observational Long-Term Study. J Am Coll Cardiol. 1998;31:1252–7.

22. Gordon DJ, Rifkind BM. High-density lipoprotein – the clinical implications of recent studies. N Engl J Med. 1989;321(19):1311–6.

23. Stern MP, Patterson JK, Haffner SM, Hazuda HP, Mitchell BD. Lack of awareness and treatment of hyperlipidemia in type 2 diabetes: A community survey. JAMA. 1989;262:360–4.

24. Stern MP, Haffner SM. Dyslipidemia in type 2 diabetes: Implications for therapeutic intervention. Diabetes Care. 1991;14:1144–59.

25. Ginsberg HN. Lipoprotein physiology in the non-diabetic state: Relationship to atherogenesis. Diabetes Care. 1991;14:1144–59.

26. Taskinen MR. Hyperlipidemia in diabetes. Baillieres Clin Endocrinol Metab. 1990;4:743–75.

27. Miller NE. Associations of HDL subclasses and apolipoproteins with ischemic heart disease. Am Heart J. 1987;113:589–97.

28. Robinson D, Ferns GA, Bevan EA, et al. HDL subfractions and coronary risk factors in normal men. Arteriosclerosis. 1987;7:341–6.

29. Scandinavian Simvastatin Survival Study Group. Randomised trial of cholesterol lowering in 4,444 patients with coronary heart disease: The 4S study. Lancet. 1994;344:1383–9.

30. Sacks FM, Pfeffer MA, Moye LA, et al. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels: The CARE trial. N Engl J Med. 1996;335:1001–9.

31. Downs JR, Clearfield M, Weis S, et al. Primary prevention of acute coronary events with lovastatin in men and women with average cholesterol levels: AFCAPS/TexCAPS results. JAMA. 1998;279:1615–22.

32. Tonkin AM, Colquhoun D, Emberson J, et al. Effects of pravastatin in 3,260 patients with unstable angina: Results from the LIPID study. Lancet. 2000;356:1871–5.

33. Cannon CP, Braunwald E, McCabe CH, et al. Intensive vs moderate lipid lowering with statins after acute coronary syndromes. N Engl J Med. 2004;350:1495–504.

34. LaRosa JC, Grundy SM, Waters DD, et al. Intensive lipid lowering with atorvastatin in patients with stable coronary disease. N Engl J Med. 2005;352:1425–35.

35. Shepherd J, Cobbe SM, Ford I, et al. Prevention of coronary heart disease with pravastatin in men with hypercholesterolemia. N Engl J Med. 1995;333:1301–7.

36. Sever PS, Dahlöf B, Poulter NR, et al. Prevention of coronary and stroke events with atorvastatin in hypertensive patients in ASCOT-LLA. Lancet. 2003;361:1149–58.

37. Abourbih S, Filion KB, Joseph L, et al. Effect of fibrates on lipid profiles and cardiovascular outcomes: A systematic review. Am J Med. 2009;122(10):962.e1–e8.

38. Gaziano JM, Hennekens CH, Satterfield S, et al. Clinical utility of lipid and lipoprotein levels during hospitalization for acute myocardial infarction. Vasc Med. 1999;4:227–31.

39. Jeppesen J. Triglycerides, HDL cholesterol, and risk of ischemic heart disease: Insights from the Copenhagen Male Study. Circ J. 2003;1(1):33–53.

40. Tan WA. Unstable Angina [Internet]. eMedicine. Available from: http://www.emedicine.com/med/byname/unstable-angina.htm

41. Lemieux I, Couillard C, Cantin B, et al. Total cholesterol/HDL ratio vs LDL/HDL ratio as indices of IHD risk in men: The Quebec Cardiovascular Study. Arch Intern Med. 2001;161:2685–92.

42. Meinrad G. Role of platelets in coronary thrombosis and reperfusion. Cardiovasc Res. 2004;61:498–51.

43. MedicineNet. Unstable angina [Internet]. Available from: http://www.medicinenet.com/script/main/art.asp?articlekey=379

44. Zevitz ME. Myocardial Ischemia [Internet]. eMedicine. Available from: http://www.emedicine.com/med/byname/Myocardial-Ischemia.htm

45. National Heart, Lung, and Blood Institute. What is Coronary Artery Disease? [Internet]. Available from: http://www.nhlbi.nih.gov/health/dci/Diseases/Cad/CAD_WhatIs.html

46. Stocker R, Yamamoto Y, McDonagh AF, Glazer AN, Ames BN. Bilirubin is an antioxidant of possible physiological importance. Science. 1987;235:1043–6.

47. The Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNCV). The fifth report. Arch Intern Med. 1993;153:154–83.

48. Hopkins PN, Wu LL, Hunt SC, Brinton EA. Plasma triglycerides and type III hyperlipidemia are independently associated with premature familial coronary artery disease. J Am Coll Cardiol. 2005;45(7):1003–12.

49. Rao N, Sastry N. Serum cholesterol levels of males and females in different age groups in South India. Am J Clin Nutr. 1980;33:181–2.

50. Kaneda H, Ohno M, Taguchi J, Hashimoto H, Ogasawara T, et al. Heme oxygenase-1 gene promoter polymorphism is associated with coronary artery disease in Japanese patients with coronary risk factors. Arterioscler Thromb Vasc Biol. 2002;22:1680–5.

51. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: An update. J Am Coll Cardiol. 2004;43:1731–7.

52. Schwertner HA. Association of smoking and low serum bilirubin antioxidant concentrations. Atherosclerosis. 1998;136:383–7.

53. Al-Refaee SA, Al-Hazzaa HM. Physical activity profile of adult males in Riyadh City. Saudi Med J. 2001;22(9):784–9.

54. Nishigaki I, Hagihara M, Tsunekawa H, Maseki M, Yagi K. Lipid peroxide levels of serum lipoprotein fractions in diabetic patients. Biochem Med. 1981;25:373–8.

55. Bierman EL. Atherogenesis in diabetes. Arterioscler Thromb. 1992;12:647–56.

56. Eknoyan G. Adolphe Quetelet – the average man and indices of obesity. Nephrol Dial Transplant. 2008;23(1):47–51.

57. Wikimedia Commons. Body mass index chart [Internet]. Available from: http://upload.wikimedia.org/wikipedia/commons/thumb/e/e9/Body_mass_index_chart.svg/400px-Body_mass_index_chart.svg.png

58. Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. Circulation. 1998;97:1837–47.

59. Barrett-Connor E, Bush TL. Estrogen and coronary heart disease in women. JAMA. 1991;265:1861–7.

60. Hammoudeh J, Izraiq M, Al-Mousa E, Tarawneh H, Elharassis A, Mahadeen Z, et al. Serum lipid profiles with and without CAD: Jordan Hyperlipidemia and Related Targets Study (JoHARTS-1). East Mediterr Health J. 2008;14(1):1–10.

61. Castelli WP, Garrison RJ, Wilson PW, Abbott RD, Kalousdian S, Kannel WB. Incidence of coronary heart disease and lipoprotein cholesterol levels: The Framingham Study. JAMA. 1986;256(20):2835–8.

62. Sharrett AR, Ballantyne CM, Coady SA, Heiss G, Sorlie PD, Catellier DJ, et al. Coronary heart disease prediction from lipoprotein cholesterol levels, triglycerides, lipoprotein(a), apolipoproteins A-I and B, and HDL density subfractions: The ARIC Study. Circulation. 2001;104:1108–13.

63. Morales KH, Wittink MN, Datto CJ, DiFilippo S, Cary M, Ten Have T, et al. Simvastatin causes changes in affective processes in elderly volunteers. J Am Geriatr Soc. 2006;54(1):70–6.

64. Britannica [Internet]. Media asset. Available from: http://media-2.web.britannica.com/eb-media/41/96841-004-065B01D0.jpg

65. Jabara R, Namouz S, Kark JD, Levine J. Risk characteristics of Arab and Jewish women with coronary heart disease in Jerusalem. IMAJ. 2007;9(4):316–20.

66. Satoh H, Nishino T, Tomita K, Tsutsui H. Fasting triglyceride is a significant risk factor for coronary artery disease in middle-aged Japanese men. Circ J. 2006;70:227–31.

67. Rafiei M, Boshtam M, Sarraf-Zadegan N. Lipid profiles in the Isfahan population: An Isfahan cardiovascular disease risk factor assay, 1994. East Mediterr Health J. 1999;5(4):766–77.

68. EUROASPIRE II Study Group. Lifestyle and risk factor management and use of drug therapies in coronary patients from 15 countries: Principal results from the EUROASPIRE II Euro Heart Survey Programme. Eur Heart J. 2001;22:554–72.

69. Haddad FH, et al. Lipid profile in patients with coronary artery disease. Saudi Med J. 2002;23(9):1054–8.

70. Khoja SM, et al. Plasma lipid levels of a selected Saudi Arabian population in the Western region. Saudi Med J. 1993;14(4):315–21.

71. Sempos CT, et al. Prevalence of high blood cholesterol among US adults. JAMA. 1993;269:3009–14.

72. Viswanathan M, Raj D, Subramaniam R, Gopal P. Prevalence of coronary artery disease and its relationship to lipids in a selected population in South

India: The Chennai Urban Population Study (CUPS No. 5). J Am Coll Cardiol. 2001;38:682–7.

73. Khwaiter SH. Risk factors associated with coronary artery disease in Gaza [MD thesis]. Gaza: The Islamic University of Gaza; 2009.