

Study of Lipid Profile and Its Correlation with Coronary Angiogram Finding in a Tertiary Care Hospital in an Urban Setting

Afshan Ara^{1,*}, Twinkle²

Abstract

Coronary artery disease is a major global health concern caused by the constriction or blockage of the coronary arteries, which can result in serious cardiovascular events. This study looks at the relationship between lipid profiles and the severity of coronary artery disease as measured by coronary angiography in patients in a tertiary care hospital. The study focuses on dyslipidemia as a major risk factor for coronary artery disease, emphasizing the importance of efficient screening and management measures. The study intends to assess the predictive usefulness of lipid fractions, such as low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides, in terms of the level of morphological impairment detected in coronary angiograms. Furthermore, the study investigates advances in non-invasive imaging techniques, particularly coronary computed tomography angiography, which has shown promise in diagnosing coronary artery disease with high accuracy. The coronary artery disease-RADS classification system is intended to standardize reporting and increase communication among healthcare practitioners, resulting in better patient management. Data were gathered using structured interviews and lipid profile tests to ensure ethical compliance and patient anonymity. The findings indicate that lipid profile components can be useful markers for diagnosing coronary artery disease severity, potentially guiding clinical decisions about invasive treatments. This study emphasizes the need to incorporate lipid profiles into routine examinations of coronary artery disease patients and calls for uniform reporting standards in coronary computed tomography angiography to improve therapeutic results. Overall, the study adds to the expanding body of evidence that supports targeted therapies for people at risk of coronary artery disease.

Keywords: Coronary artery disease (CAD), lipid profile, dyslipidemia, coronary angiography, LDL cholesterol, coronary computed tomography angiography (CCTA), risk factors

INTRODUCTION

Coronary artery disease (CAD), also referred to as coronary heart disease or ischemic heart disease, is a prevalent and serious condition affecting the heart. It arises when the coronary arteries, which provide oxygen and essential nutrients to the heart muscle, become narrowed or obstructed [1–3].

These arteries are vital for the heart's proper functioning as they deliver oxygen-rich blood. However, certain factors can lead to the buildup of plaque, consisting of cholesterol, fat, calcium, and other substances, on the artery walls. This gradual accumulation can harden the arteries over time, leading to a condition known as atherosclerosis [4, 5]. Figures 1 and 2 depict the CAD and its types.

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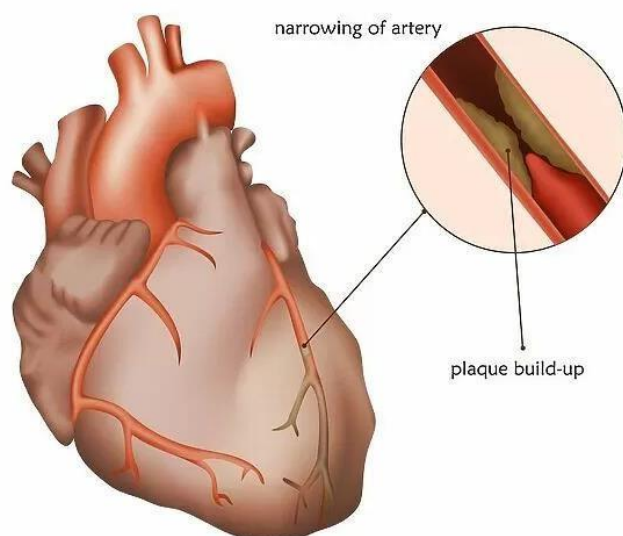


Figure 1. CAD.

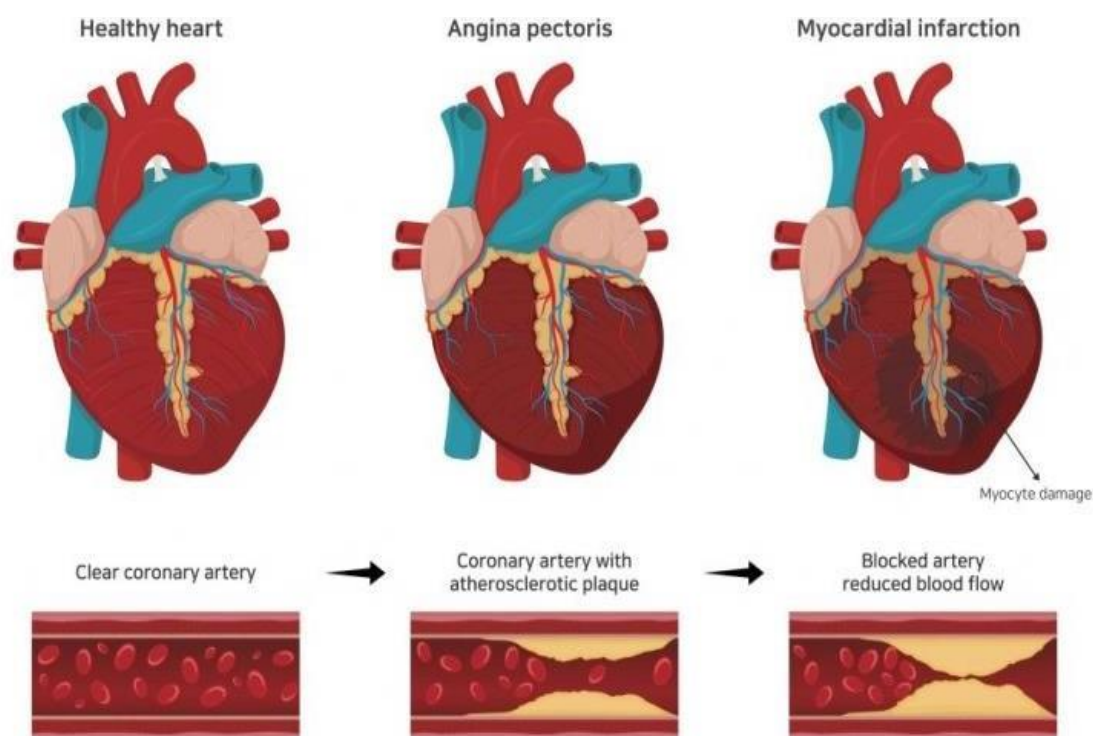


Figure 2. Types of CAD lipid profile.

A lipid profile, also known as a lipid panel or lipid profile test, is a blood test that measures various types of lipids (fats) and lipoproteins present in your bloodstream. It provides valuable information about your cholesterol levels and helps assess your risk for developing heart disease, stroke, and other cardiovascular conditions [6].

Lipid profile tests are usually performed after a period of fasting, typically for 9–12 hours, to obtain accurate results. The results of the lipid profile can help your healthcare provider assess your cardiovascular health, determine if you have abnormal lipid levels, and develop appropriate strategies to manage and reduce your risk of heart disease [7].

It's important to note that the interpretation of lipid profile results may vary depending on individual factors, such as age, gender, medical history, and the presence of other risk factors. Therefore, it is crucial to consult with a healthcare professional who can provide personalized guidance based on your specific circumstances [8–10].

TYPES

The lipid profile consists of different components that provide information about various lipids and lipoproteins present in the blood. Here are the primary types of measurements included in a typical lipid profile (Figure 3):

1. **Total Cholesterol:** This is a measurement of all the cholesterol in the bloodstream, encompassing both HDL (high-density lipoprotein) and LDL (low-density lipoprotein) cholesterol [11].
2. **LDL Cholesterol:** Known as “bad” cholesterol, LDL cholesterol can lead to plaque buildup in the arteries, which raises the risk of heart-related diseases.
3. **HDL Cholesterol:** Often referred to as “good” cholesterol, HDL helps clear excess cholesterol from the blood, transporting it to the liver for removal. Higher HDL levels are linked to a decreased risk of cardiovascular diseases [12].
4. **Triglycerides:** Triglycerides are fats present in the bloodstream that store surplus energy from food. High triglyceride levels are associated with an increased risk of heart disease [13].

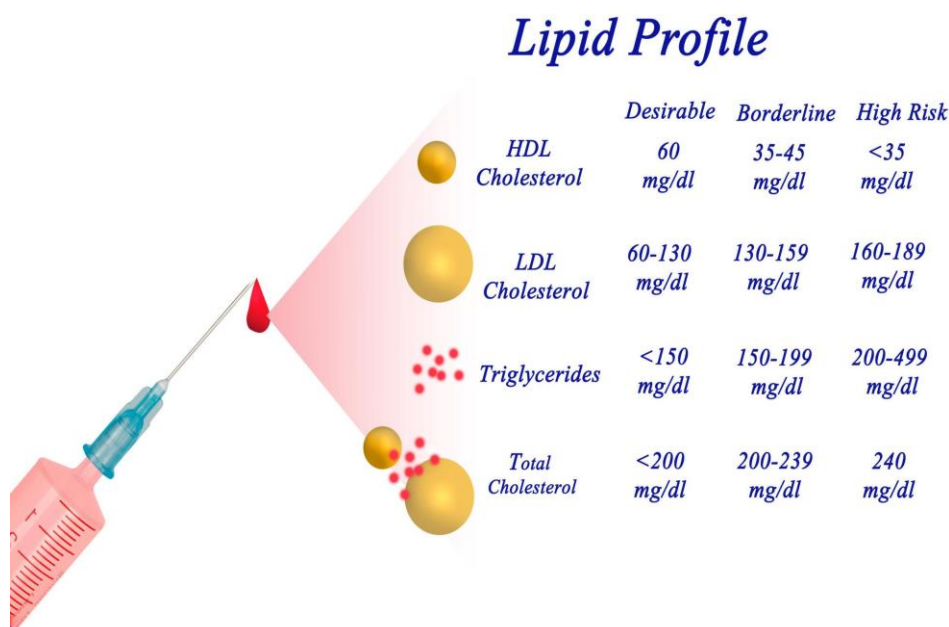


Figure 3. Different values of different subsets of lipid profile.

Effect of Lipid Profile on CAD

The lipid profile is crucial in the onset and progression of CAD, a condition marked by the narrowing or blockage of coronary arteries, which supply blood to the heart. Here is how the components of a lipid profile influence CAD.

1. **LDL Cholesterol:** High levels of LDL, often labeled as “bad” cholesterol, play a key role in atherosclerosis, where cholesterol gets deposited in artery walls, triggering inflammation and plaque formation. This can narrow the arteries, restricting blood flow to the heart and increasing the likelihood of heart attacks and other cardiovascular issues [14].
2. **HDL Cholesterol:** Elevated levels of HDL, or “good” cholesterol, are linked to a reduced risk of CAD. HDL removes excess cholesterol from the bloodstream, transporting it to the liver for elimination. Its anti-inflammatory and antioxidant properties may protect against plaque formation, thereby lowering the risk of CAD [15].

3. *Triglycerides*: High levels of triglycerides, a type of fat in the blood, are associated with an elevated risk of CAD. Increased triglycerides are often seen alongside risk factors like obesity, insulin resistance, and metabolic syndrome. These fats contribute to atherosclerosis and may also affect CAD risk by influencing other lipid profile elements [16].
4. *Total Cholesterol*: Elevated total cholesterol, mainly due to high LDL, contributes to the development of CAD. Since total cholesterol includes both LDL and HDL, an imbalance can result in plaque buildup in the coronary arteries [17].
5. *Non-HDL Cholesterol*: Non-HDL cholesterol, calculated by subtracting HDL from total cholesterol, includes LDL and other cholesterol-rich particles. It provides a broader assessment of all lipoproteins that can lead to CAD [18].

AIM

Study of lipid profile and its correlation with coronary angiogram finding in a tertiary care hospital in an urban setting.

OBJECTIVES

1. To determine the correlation between the lipid profile of patients with coronary angiography findings.
2. To determine effects of different subsets in lipid profile with patient outcomes and disease findings.
3. To determine the lipid profile pattern in Indian patients admitted at multispecialty hospitals.

MATERIALS AND METHODS

1. *Study Setting*: Grecian hospital, Mohali.
2. *Study Duration*: 1 year
3. *Definition of Problem*: Patient admitted to the multispecialty hospital undergoing coronary angiography and the relationship between the lipid profile patterns in Indian patients and involvement having overall effects of different subsets in lipid profile with patient outcomes and disease findings.
4. *Definition of Population*: Patient undergoing coronary angiography in a multispecialty hospital (Grecian hospital).
5. *Inclusion Criteria*: All patients undergoing coronary angiography and having their lipid profile test.
6. *Exclusion Criteria*: The patient who is diagnosed with congenital heart disease and valvular heart disease after coronary angiography should be excluded.
7. *Sample Size*: The sample size is 223 patients those are fulfilled the criteria of lipid profile of patients undergoing coronary angiography and agreed to the questionnaire along with co-morbid illness after they were admitted to our hospital, were amenable to follow-up and who gave proper consent. Initially, the sample size was more but they were excluded due to loss of data and follow-up, incomplete filling of questionnaire, or other major illness confounding research.
8. *Sample Design*: Retrospective study.
9. *Method of Data Collection*: Data were collected for all patients having coronary angiography along with lipid profiles admitted to our hospital in CCU (Coronary Care Unit) and cath lab department with the help of a standard validated questionnaire along with standard patient profile form. Those patients were discharged and had already received therapy, their previous reports were taken from MRD (Medical Record Department) using computers database and this will follow for the study duration.
10. *Study Design*: Observational study.
11. *Definition of Outcome*: To determine subsets analysis of lipid profile with patients undergoing coronary angiography in the multispecialty hospital.
12. *Schedule of Data Collection*: In Grecian multispecialty hospital patients after admission to the hospital and undergo coronary angiography.

RESEARCH METHODOLOGY

The methodology is a crucial component of any research, providing a framework for organizing the process of collecting reliable data relevant to the research problem. This section outlines the methodology used in the study, detailing the steps involved in gathering and organizing data.

Research methods refer to the techniques employed by researchers to design a study, collect, and analyze data that addresses the research question. The methodology chapter typically includes the research approach, design, variables, setting, population, sample size, sampling method, sampling criteria, development and description of tools, validity and reliability testing, pilot study, data collection process, analysis plan, and ethical considerations. The stages of the lipid profile are depicted in Table 1 and Figure 4 depicts the study design.

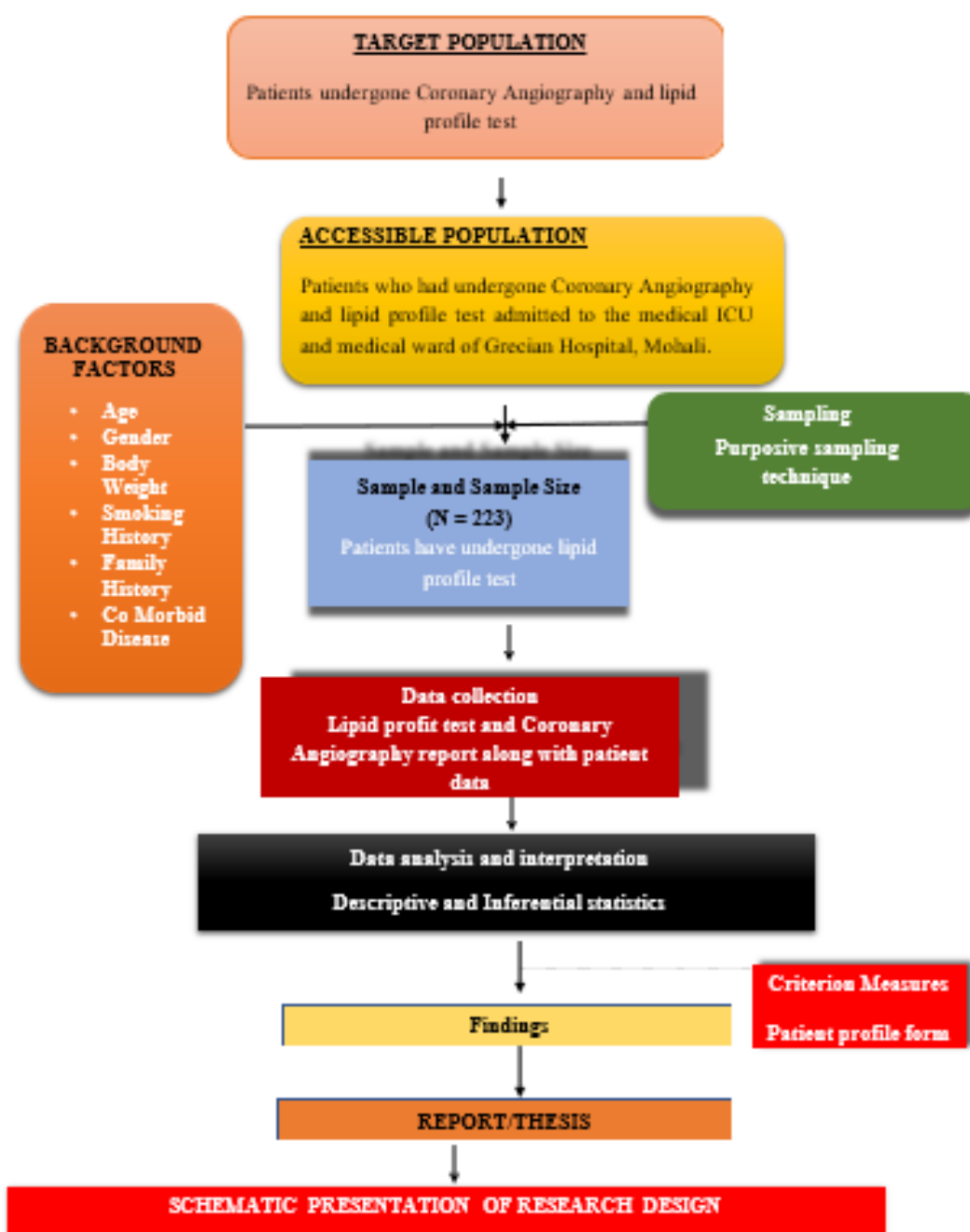


Figure 4. Study design.

Table 1. Stages of lipid profile.

Lipoprotein	Desirable	Borderline	High risk
HDL	60 mg/dl	35–45 mg/dl	<35 mg/dl
LDL	60–130 mg/dl	130–159 mg/dl	160–189 mg/dl
TGS	<150 mg/dl	150–199 mg/dl	200–499 mg/dl
S. cholesterol	<200 mg/dl	200–239 mg/dl	>240 mg/dl

Coronary Angiogram Findings

Coronary angiogram findings are used to determine different kinds of obstruction in the Coronary Artery. It helps to measure the level of CAD along with the percentage of occlusion/plaque in the vessels. Resulting in the decision of minor and severe CAD. Depending on the vessels CAD can be classified as:

- SVD: Single vessel disease.
- DVD: Double vessel disease.
- TVD: Triple vessel disease.

Patient Profile Form

It helps to collect all the data in a single form containing all the information regarding the patient selected by the criteria. It contains information including data on patients, such as demographic information, lab reports, lipid profile tests, angiogram findings, treatment/therapy, medication, and its doses.

DATA COLLECTION PROCEDURE**Phase I: Screening Phase**

The study was carried out at Grecian Hospital in Mohali over the course of one year. Official written permission was obtained from the Hospital Director via the elective coordinator. With the help of the ward sisters and from the information obtained from the patient's medical records, patients who fulfilled the sample selection criteria were selected to be placed.

Phase II: Data Collection and Implementation Phase

After identifying the samples, the purpose and procedure of data collection were explained to the patients and written consent was obtained from them. Confidentiality was ensured. The structured interview method was used to collect personal details of patients among cases and controls. Lipid profile test reports were taken from the patient's medical records with the help of the concerned ward in charge.

Phase III: Termination Phase

The tool was verified for its completion. The investigator shared his vote of thanks to the patients and the ward sister and concerned hospital authorities for permission, cooperation, and willingness to participate in the study. The patients were assured about the confidentiality of the data [19, 20].

PLAN FOR DATA ANALYSIS

Data analysis is the systematic organization and synthesis of research data and testing of the research hypothesis using the data. The data collected from the subjects were compiled and analyzed by using descriptive and inferential statistics. The following plan of analysis was developed.

- The distribution of samples according to background factors was explained by using frequency and percentage.
- Frequency and percentage were used to assess the exposure rate of lipid profile and risk level of CAD.
- Odd ratio to estimate the risk.
- Chi-square was used to associate the demographic variables and CAD.

THE DATA ANALYSIS IS PRESENTED AS FOLLOWS

- *Section I:* Information on the selected background factors of the participants.
- *Section II:* Data regarding the exposure rates of lipid profiles and the risk levels of CAD among the participants.
- *Section III:* Data on the estimated risk related to lipid profiles and CAD among the participants.
- *Section IV:* Analysis of the association between the demographic profile and the risk levels of CAD among the cases.
- *Section V:* Data on additional findings related to this study.

STUDY METHODOLOGY

In this cross-sectional study on 223 individuals who underwent elective coronary angiography, due to angina-like chest pain and/or positive treadmill exercise test or positive coronary computed tomography angiography (CCTA), in Grecian super specialty hospital over a period of one year from the beginning of July 2022 till end of May 2023.

Participants were divided according to the number of affected vessels into 3 groups; Group 1 (n =100): includes those with non-obstructive CAD; Group 2 (n = 38): includes those with single vessel disease; Group 3 (n=85) includes patients with two and three vessel diseases (multi-vessel CAD) [21].

The patients were then regrouped into four groups according to CAD presentations into chronic stable angina (n = 145), non-ST segment elevation and/or unstable angina (n = 50), and those with ST-segment elevation (n = 28). Blood samples were collected in the early morning before cardiac catheterization in the Cath lab and sent directly to the laboratory for fasting blood glucose and lipid profile measurements using an enzymatic, colorimetric method by using a COBAS Integra 400 plus analyzer and commercial kits from Roche Company to measure total cholesterol, LDL and HDL directly.

The lesion was considered significant if the luminal stenosis >50% occlusion in the left main coronary artery and/or an occlusion greater than 70% in any of the other coronary arteries were diagnosed with obstructive CAD [22].

The results were expressed as mean $\pm$ SD compared using one-way analysis of variance (ANOVA) performed by the Statistical Package for Social Science (SPSS) version. Results expressed as percentages were compared using the Chi-Square test. Statistically significant differences were considered when $P < 0.05$ [23].

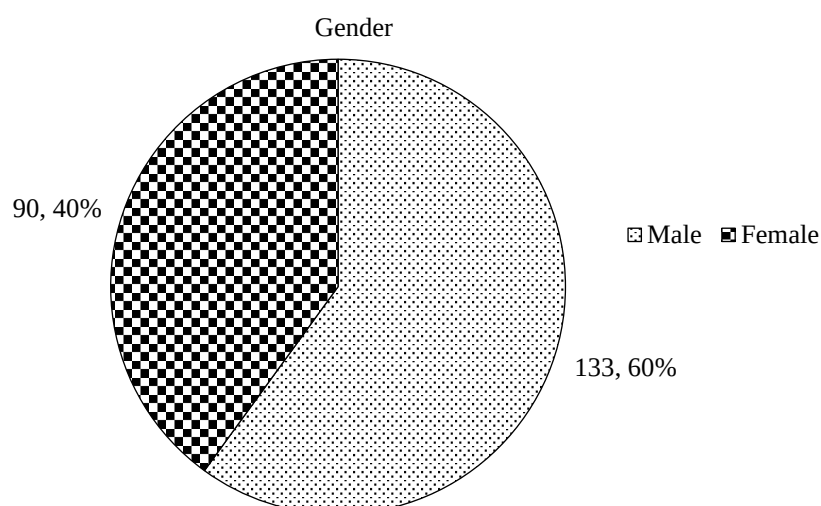


Figure 5. Frequency and percentage distribution of demographic variable gender among cases.

Frequency and percentage distribution of demographic variable gender among cases. Regarding gender, majority 133 (60%) were males and 90 (40%) were females among cases (Figure 5).

RESULTS

Table 2 shows that current smoking, diabetes mellitus, physical inactivity, family history of ischemic heart disease, and dyslipidemia are strongly associated with the severity of CAD in relation to number of affected vessels. While age, hypertension, and obesity were not related to the severity of CAD in relation to the number of affected vessels (Figures 6-8).

Table 2. Population characteristics according to number of vessels.

S.N.	Variables	No Obstruction (n = 100)	One Vessel Disease (n = 38)	Multi-Vessel Disease (n = 85)	P-value
1.	Age (mean $\pm$ SD)	58.1 $\pm$ 7.6	55.5 $\pm$ 9.2	56.4 $\pm$ 10.4	≥ 0.05
2.	Sex (%)				
3.	Male	59 (59%)	26 (68.4%)	48 (56.5%)	≥ 0.05
4.	Female	41 (41%)	12 (31.6%)	37 (43.5%)	
5.	BMI (kg/m ²) (mean $\pm$ SD)	28 $\pm$ 4.2	30.1 $\pm$ 4	30.1 $\pm$ 5.5	≥ 0.05
6.	Normal weight (%)	11 (11%)	6 (15.7%)	21 (24.7%)	≥ 0.05
7.	Overweight (%)	47 (47%)	12 (31.6%)	27 (31.8%) ^a	≥ 0.05
8.	Obese (%)	42 (42%)	20 (52.6%)	37 (43.5%)	≥ 0.05
9.	Ejection fraction (mean $\pm$ SD)	55.7 $\pm$ 10.9	58.1 $\pm$ 8.7	54.9 $\pm$ 9.1	≥ 0.05
10.	Current smoker	15 (15%)	12 (31.6%) ^a	40 (47.1%) ^{ab}	<0.0001
11.	Ex-smoker	23 (23%)	4 (10.5%)	24 (28.2%) ^b	≥ 0.05
12.	Never smoke	62 (62%)	22 (57.9%) ^a	21 (24.7%) ^{ab}	<0.0001
13.	Diabetes mellitus	29 (29%)	18 (47.4%) ^a	40 (47.1%) ^a	0.022
14.	Hypertension	55 (55%)	19 (50%)	44 (51.8%)	≥ 0.05
15.	Dyslipidemia	37 (37%)	24 (63.2%) ^a	57 (67.1%) ^{ab}	<0.0001
16.	Family history of IHD	24 (24%)	15 (39.5%) ^a	44 (51.8%) ^{ab}	0.0005
17.	Sedentary lifestyle	32 (32%)	16 (42.1%) ^a	53 (62.4%) ^{ab}	0.0002

Table 3 shows total cholesterol, LDL and non-HDL cholesterol were found strongly associated with the severity of CAD while triglycerides, total cholesterol/HDL ratio, and LDL/HDL ratio were not related with the severity of CAD in relation to the number of vessels occluded.

Table 4 shows that patients with ST-segment elevation myocardial infarction were significantly younger than other presentations of CAD.

Diabetes mellitus, dyslipidemia, and physical inactivity were strongly associated with severe presentation of CAD like ST segment elevation and non-ST segment elevation and/or unstable angina. Other risk factors including obesity, smoking, hypertension, and a family history of ischemic heart disease showed no significant difference in the presentation of CAD. Dyslipidemia was strongly associated with non-ST segment elevation myocardial infarction and/or unstable angina.

Table 5 shows that total cholesterol, elevated triglycerides, and non-HDL cholesterol were found strongly associated with more severe forms of CAD.

Table 3. Association of lipid profile and number of vessels occluded.

Lipid Profile (mean ± SD)	Non-Obstruction (n = 100)	One Vessel Disease (n = 38)	Multi-Vessel Disease (n = 85)	P-value
Total serum cholesterol (mg/dl)	175.2 ± 44.3	191.2 ± 40.8	201.7 ± 53. ^a	0.001
LDL cholesterol (mg/dl)	110.1 ± 29.9	114 ± 34.3	127.1 ± 45.7 ^a	0.008
HDL cholesterol (mg/dl)	44.4 ± 9.6	48.3 ± 13.1 ^a	47.6 ± 15.7 ^a	≥0.05
Triglycerides (mg/dl)	152.2 ± 66.4	158.2 ± 82.8	66.3 ± 81.5	≥0.05
Non-HDL (mg/dl)	132.9 ± 44.3	141.9 ± 38.2	153.1 ± 53.1 ^a	0.016
TC-HDL ratio	4.4 ± 1.5	4.1 ± 1.2	4.7 ± 2.1	≥0.05
LDL-HDL ratio	2.8 ± 1.1	2.4 ± 0.9	2.9 ± 1.4 ^b	≥0.05

a. Significant at $P < 0.05$ as compared with non-obstruction values.

b. Significant at $P < 0.05$ as compared with mild obstruction values.

Table 4. Characteristics of population and the presentation of CAD.

S.N.	Variables	Stable Angina (n = 145)	ACS-NSTEMI (n = 50)	STEMI (n = 28)	P-value
1.	Age (mean ± SD)	58 ± 8.8	56 ± 9.9	53.4 ± 7.8 ^a	0.031
2.	Gender (%)				
3.	Male	85 (58.6%)	31 (62%)	17 (60.7%)	≥0.05
4.	Female	60 (41.4%)	19 (38%)	11 (39.3%)	
5.	BMI (kg/m ²) (mean ± SD)	29.1 ± 4.9	28.7 ± 5.3	30.5 ± 4	≥0.05
6.	Normal weight (%)	26 (17.9%)	10 (20%)	2 (7.1%)	≥0.05
7.	Overweight (%)	59 (40.7%)	17 (34%)	10 (35.7%)	≥0.05
8.	Obese (%)	60 (41.4%)	23 (46%)	16 (57.1%)	≥0.05
9.	Ejection fraction (mean ± SD)	56.3 ± 10.2	56.1 ± 9.6	52.9 ± 8.9	≥0.05
	Smoking history				
10.	Current smoker	38 (26.2%)	17 (34%)	12 (42.9%)	≥0.05
11.	Former smoker	29 (20%)	15 (30%)	7 (25%)	≥0.05
12.	Never smoke	78 (53.8%)	18 (36%) ^a	9 (32.1%) ^{ab}	0.0224
13.	Diabetes mellitus	51 (35.2%)	21 (42%)	15 (53.6%)	0.0167
14.	Hypertension	80 (55.2%)	25 (50%)	13 (46.4%)	0.625
15.	Dyslipidemia	70 (48.3%)	33 (66%) ^a	15 (53.6%)	0.03
16.	Family history of IHD	49 (33.8%)	20 (40%)	14 (50%)	≥0.05
17.	Sedentary lifestyle	55 (37.9%)	30 (60%) ^a	16 (57.1%) ^{ab}	0.0004

a. Significant at $P < 0.05$ as compared with angina.

b. Significant at $P < 0.05$ as compared with ACS value.

Table 6 shows that the most common vessel involved in all subgroups was LAD (32.7%) followed by LCX (27.2%), then RCA (24.1%), and lastly LMS (16%).

Table 5. Association between lipid profile and severity of CAD presentation.

Lipid Profile (mean ± SD)	Stable Angina (n = 145)	NSTEMI &/or UA (n = 50)	STEMI (n = 28)	P-value
Total serum cholesterol (mg/dl)	183.7 ± 46.1	200.6 ± 53.1 ^a	188 ± 50.7	0.02
LDL cholesterol (mg/dl)	110.5 ± 30.4	115.6 ± 44.9	121.5 ± 40.1	≥0.05
HDL cholesterol (mg/dl)	45.4 ± 12.5	45.9 ± 14.9	48.7 ± 13.3	≥0.05
Triglycerides (mg/dl)	155.2 ± 72.3	148.9 ± 53.9	191.5 ± 109.5	0.042
Non-HDL (mg/Dl)	138.38 ± 45.1	154.7 ± 50.1 ^a	139.3 ± 54.1	0.01
TC-HDL Ratio	4.4 ± 1.8	4.7 ± 1.5	4.2 ± 1.8	≥0.05
LDL-HDL Ratio	2.8 ± 1.2	2.8 ± 1.4	2.6 ± 1	≥0.05

Note: a: Significant at $P < 0.05$ as compared with stable angina values.

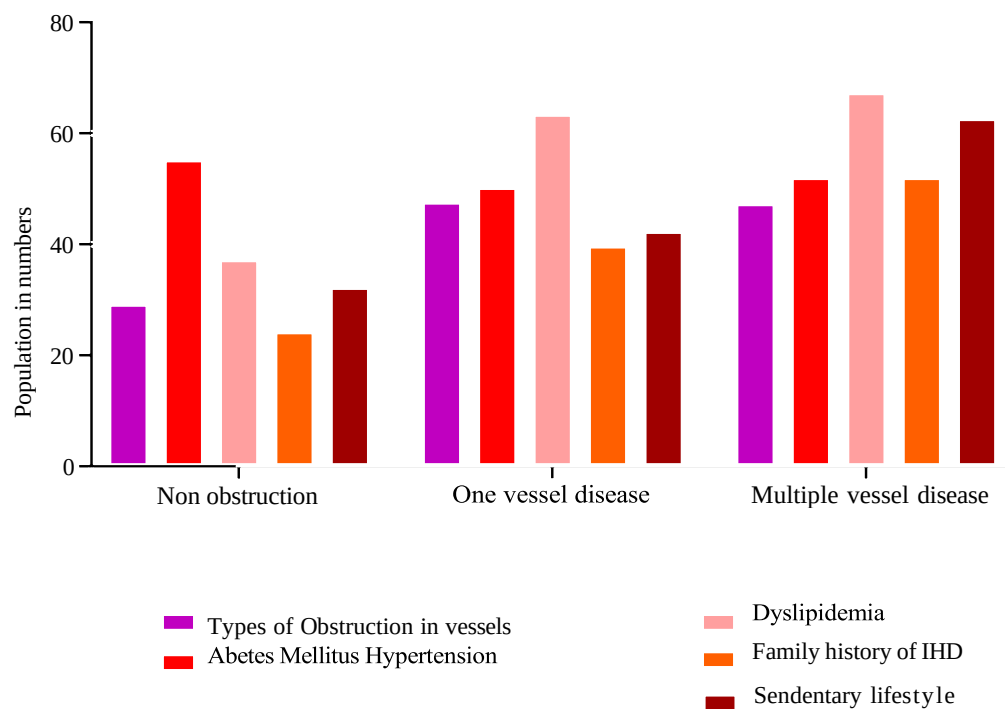


Figure 6. Representation of types of obstruction in vessels of a percentage of disease in dm, HTN, dyslipidemia, family history of IHD, and sedentary lifestyle.

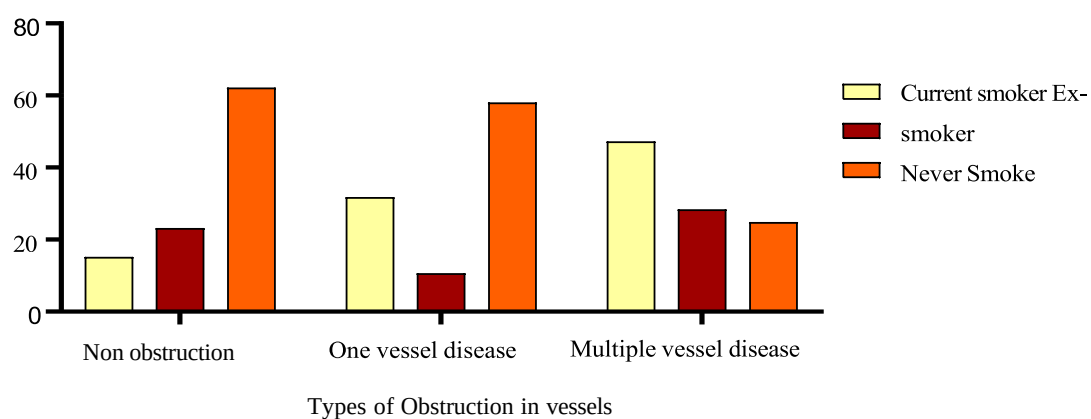


Figure 7. Representation of types of obstruction in vessels of percentage of smokers in current smokers, ex-smoker, and never smokers.

Table 6. Types of coronary vessel involved.

Types of Coronary Vessel Involved	Overall Ratio (n = 275 Records)
LCMA	41 (16%)
LAD	84 (32.7%)
CFX	70 (27.2%)
RCA	62 (24.1%)

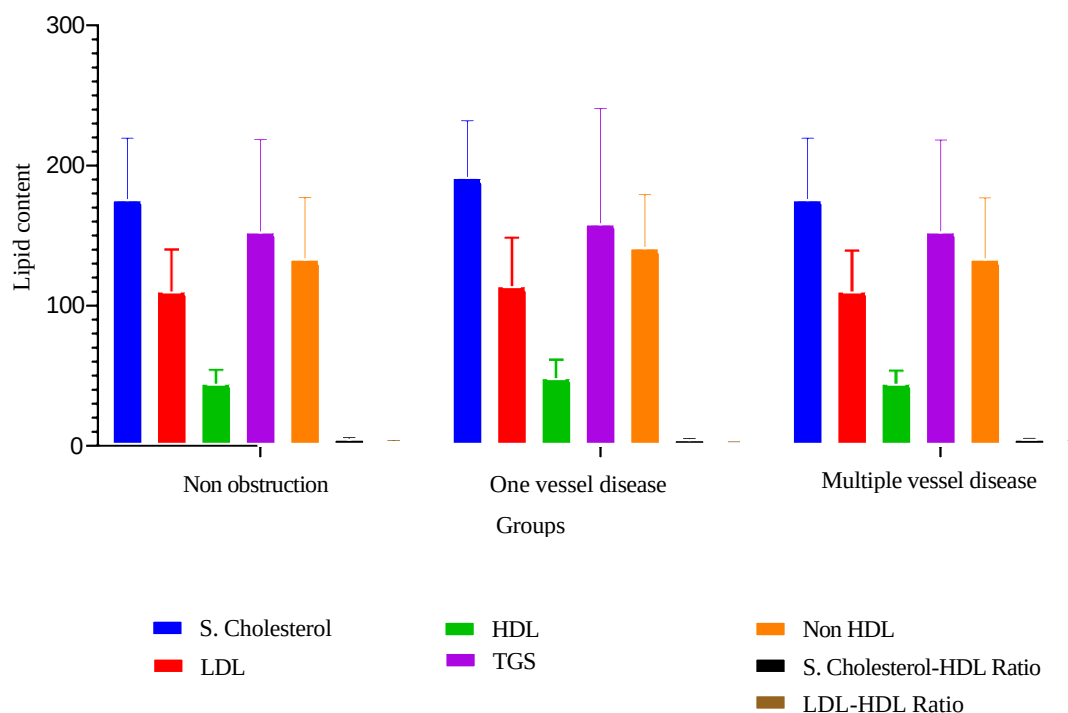


Figure 8. Representation of types of obstruction in vessels of lipid profile.

DISCUSSION

It is well-known that dyslipidemia is a major risk factor for CAD. Research on the significance of lipoprotein levels as indicators of CAD severity remains limited in the literature. It is suggested that if a specific lipid fraction can predict the extent of anatomical damage seen in coronary angiography, it could influence decisions regarding the use of invasive diagnostic procedures in CAD patients [24].

Age, in this study; did not significantly affect the number of vessels occluded but; disagreed with the study in which patients with multi-vessels disease were older. In terms of gender, there was no significant difference in the number of occluded vessels between men and women. This result contrasts with findings from studies on obesity in this study. This result could be explained by the greater likelihood that physicians would refer to obese patients for coronary angiography at an earlier stage of CAD [25].

Current smoking is strongly associated with the severity of CAD (number of affected vessels), this result is consistent with the study. This can largely be attributed to vascular endothelial dysfunction and the activation of the sympathetic nervous system caused by cigarette smoking, which may lead to an insufficient or inadequate increase in coronary blood flow in response to heightened myocardial demand. A significantly higher proportion ($p < 0.05$) of multi-vessel CAD patients were found to have diabetes (47.1%), suggesting that diabetes may be linked to the severity of CAD, a finding consistent with previous research. Patients with DM experienced more multi-vessel disease than nondiabetic patients and this finding is consistent. This may be mostly explained by coronary endothelial dysfunction and atherosclerotic processes induced by glucose toxicity, inflammatory, and immune-mediated mechanisms [26–28].

Hypertension was not significantly associated with CAD severity, and this comes. Dyslipidemia was strongly associated with an elevated occurrence of obstructive CAD and was associated with its severity, which is consistent with Khashayer (2010) who stated that “dyslipidemia was more prevalent among those with more severe coronary diseases”. A family history of CAD was significantly

associated with multi-vessel disease, and similar results were revealed. Physical inactivity was associated with more severe CAD, which is similar to that published in AHA statement about the role of “Exercise and Physical Activity in the Prevention and Treatment of Atherosclerotic Cardiovascular Disease”. The average age of patients with STEMI was significantly lower than patients with other presentations of CAD like that published by Mohammad. Gender was unrelated to the presentation of CAD while obesity had a higher percentage in ACS patients but it was not significant and these were consistent with Ameen, et al. study [29, 30].

CONCLUSIONS

Total cholesterol, LDL cholesterol, and non-HDL cholesterol were identified as indicators of CAD severity concerning the number of affected vessels. Dyslipidemia showed a strong correlation with NSTEMI and/or unstable angina. Additionally, total cholesterol, triglycerides, and non-HDL cholesterol levels were elevated in patients with acute coronary syndrome compared to those with stable angina.

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