Journal of Chemical Health Risks

www.jchr.org

JCHR (2026) 16(1), 1325-1336 | ISSN:2251-6727

Correlation of Lipid Sub-Fractions in Angiographically Proven Coronary Artery Disease Patients.

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(Received: 05 November 2025 Revised: 15 December 2025 Accepted: 23 January 2026)

KEYWORDS

Lipid Subfractions, Apo-Lipoprotein A(APO A), Apo-lipoprotein B(APO B), coronary artery disease patients, angiography.

ABSTRACT:

Background:- Aging is marked by a decrease in biological functions and lesser cellular resistance to stress. It is known that longevity is a characteristic that is influenced by severalgenes, among that is Apo-lipoprotein (APO) genes. An Apo protein has impact on processes relatedto cellular stress response possibly, due to connections among mitochondrial functions, endoplasmicreticulum stress and immuneresponse. This is the reason that Apo-proteins may be risk factors for development of certain diseases related to atherosclerosis, notablycoronary artery disease. Therefore, we have intended to carry out this study of correlation of Apo A1/Apo B for estimating atherogenecity in relation to its ratio with severity of coronaryartery disease.

Objective:- Study of correlation of lipid sub-fractions with severity of coronary artery disease in angiographically proven cases.

Methodology:- Prospective Observational Study, 126 Patients, conducted at MGM Medical College and Hospital, Chh. Sambhajinagar.

Results:- Lipid profile analysis revealed higher levels of total cholesterol, LDL, triglycerides,and APO Bin DVD and TVD compared to SVD, with significant differences noted.

Conclusion: Elevated levels of total cholesterol, LDL, triglycerides, APOB were significantly associated with more severe forms of CAD, highlighting these lipid sub-fractions as key markers for disease severity. The finding sunder scores the importance of managing lipid levels and comorbid conditions in CAD patients to mitigate disease progression.

INTRODUCTION:-

Coronary artery disease (CAD) is one of the major cardiovascular diseases (CVD) affectingthe global human population. [1]. An increase in coronary risk factors, such as central obesity, diabetes ,hypertension ,atherogenic dyslipidemia, smoking, and physical inactivity, contributes to the rise in thisdisease.[2]. Recent research now includes new factors like Apolipoprotein A1(ApoA1)and Apolipoprotein B (ApoB) inassessing risks, along with traditional ones.[3].According to themeta-analysis as well as epidemiological and clinical studies, individuals with low plasma levels of high-density lipoprotein cholesterol (HDL-C) are at anhigh risk ofexperiencing majorcoronaryevents.[4]

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The major protein component of HDL is Apo A1 [5] and it accounts for approximately 70%of total HDL protein. Apo A1 activates lecithin cholesterol acyltransferase (LCAT) whichcatalyses the esterification of cholesterol. This esterified cholesterol can then be transportedto the liver, metabolized and excreted. Individuals with atherosclerotic vascular changes frequently exhibit decreased levels of ApoA1. Eveninnormal ApoBconcentration, decrease level of Apo A1 remains a risk factor for artehrosclerosis. The second main HDLapolipoprotein is Apo A-IIwhich makes up 15%–20% ofthe total HDLprotein.[6]

Furthermore, Apo B is the primary apolipoprotein and is the carrier for the lipids such aschylomicrons, low-densitylipo protein (LDL), verylow-densitylipo protein (VLDL), intermediated ensitylipoprotein (IDL), and lipoprotein (a). There are two circulating forms of Apo B which includes Apo B48 (from the small intestine) and Apo B100 (from the liver) and same gene codes for both ApoB48 an ApoB 100. [7] Apo B is the main protein of low density lipoprotein (LDL). Thus, ApoB is a more accurate indicator of the quantity of LDLpartic les than LDL cholesterol.[8]

In 2015, The National Lipid Association concluded that, high levels of ApoB – contain inglipoproteins, specifically non-HDL cholesteroland its main component, LDL cholesterol, are a primary cause of atherosclerosis, which is the key underlying process that eventually leads to clinical CVD events like myocardial infarction and stroke.[9,10]

For the diagnostic purpose of CAD, invasive coronary angiography plays a crucial role.Coronary angiography involves visualization of the coronary anatomy using fluoroscopy. This is done by injecting contrast media directly into the epicardial coronary arteries via a catheter inserted from aperipheral arteryto the aortic root and then into the coronaryostia.

Furthermore, aging is marked by a decrease in biological functions and lesser cellular resistance to stress. It is known that longevity is a characteristic that is influenced by several genes, among that is Apo-lipoprotein genes. An Apo protein has impact on processes relatedto cellular stress response possibly, due to connections among mitochondrial functions, endoplasmicreticulum stress and immune response.This is the reason that Apo-proteins may be risk factors for development of certain diseases related to atherosclerosis, notablycoronary artery disease. Therefore, we have intended to carry out this study of correlation ofApo A1/Apo B for estimating atherogenecity in relation with severity of coronary artery disease.

AIM

To study the correlation of lipid sub-fractions in angiographically proven coronary artery disease patients.

OBJECTIVES

Study of correlation of lipid sub-fractions with severity of coronary artery disease in angiographically proven cases undertaking parameters:

a. Total serum cholesterol

b. Total serum triglycerides

c. Serum HDL, LDL,VLDL

d. APOB and APOA1.

2.To compare the correlation of incidence of younger age CAD (<40years) and old age CAD (>40years) with Lipid-Sub fractions.

Material and Methods:

This Prospective observational study was conducted at MGM Medical College and Hospital, Chh. Sambhajinagar, on 126 angiographically proven Coronary Artery Disease (CAD) patients, divided into two groups: individuals under the ageof 40 and those over 40 years. CAD was deemed significant when coronary blockageexceeded 50%. Severity was categorized as Single Vessel Disease (SVD), Double Vessel Disease (DVD), or Triple Vessel Disease (TVD), reflecting the extent of arterial involvement. This approach ensured a comprehensive analysis of CAD across different age brackets and severity levels, contributing valuable insights into its manifestation and progression. Study included Angiographically proven coronary artery disease patients above 18 years and Patients with Structural heart disease. (Rheumatic heart disease, Valvular Heart disease, Cardiomyopathies, Pericardial disorders, Congenital heart disorders etc)

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JCHR (2026) 16(1), 1325-1336 | ISSN:2251-6727

and chronically on Lipid lowering drugs (Statins, Fibrates and Herbal Preparations) were excluded.

Fasting venous blood was obtained from each patient, was collected and lipo protein sub fractions were measured. Concentrations of VLDL, LDL, triglycerides, HDL, cholesterol, apolipoprotein AI (apo AI) and apolipoprotein B (apo B) were measured by VITROSS5600 EQUIPMENT. Institutional Ethics Committee approval and written informed consent from patients were obtained.

Results:

Table-1:Demographic characteristics of participants

Variables	**Mean±SD**	
Age	56.75 ± 16.11	
Gender	**Noof cases**	**Percentage**
Female	35	27.78%
Male	91	72.22%
Total	126	100.00%

The study involved 126 participants with angiographically proven coronary artery disease, comprising 35 females (27.78%) and 91 males (72.22%). The average age of the participants was 56.75 years, with a standard deviation of16.11years.

Table-2:Age distribution among all groups

Age	**SVD**	**DVD**	**TVD**	**P-value**
Mean±SD	46.21 ± 13.90	60.52 ± 14.71	65.35 ± 13.82	<0.0001

The mean age for those with SVD was 46.21 years (SD ± 13.90), for DVD it was 60.52years (SD ± 14.71), and for TVD it was 65.35 years (SD ± 13.82). The differences in age across these groups were statistically significant, with a p-value of less than 0.0001, indicating that older ageis associated with greater disease severity.

Table-3:Genderdistributionamongallgroups

Gender	**SVD** **No of cases (%)**	**DVD** **No of cases (%)**	**TVD** **No of cases (%)**	**Chi-square**	**P-value**
Female	12(28.57%)	17(29.31%)	6(23.08%)	0.37	0.54
Male	30(71.43%)	41(70.69%)	20(76.92%)		

Among those with SVD, 12 were female (28.57%) and 30 were male (71.43%). For DVD,17 were female (29.31%) and 41 were male (70.69%). In the TVD group, 6 were female (23.08%) and 20 were male (76.92%). The differences in gender distribution across these groups were not statistically significant, with a chi-square value of 0.37 and a p-value of0.54.

Table-4: Distribution of clinical parameters among all groups

Variables	**SVD** **No of cases (%)**	**DVD** **No of cases (%)**	**TVD** **No of cases (%)**	**P-value**
Height(cm)	166.95 ± 9.44	166.08 ± 8.08	165.62 ± 9.23	0.81

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Weight(Kg)	66.17± 8.28	68.53 ± 11.05	76.92 ± 16.55	0.001
BMI(Kg/m2)	23.57± 3.04	24.72 ± 3.22	28.68 ± 7.39	<0.0001

Height did not differ significantly across coronary artery disease severity groups (p = 0.81). However, weight (p = 0.001) and BMI (p < 0.0001) were significantly higher in participants with more severe disease, with those having TVD showing the highest values (Weight:76.92± 16.55 kg,BMI: 28.68± 7.39 kg/m²).

Table-5: Distribution according to chief complaints among all groups

Chief complaints	SVD	DVD	TVD	Chi-square	P-value
	No of cases (%)	No of cases (%)	No of cases (%)		
Breathlessness	9(21.43%)	11(18.97%)	7(26.92%)	2.75	0.098
chestpain	25(59.22%)	35(60.34%)	16(61.54%)		
Chest pain and breathlessness	2(4.76%)	5(8.62%)	2(7.69%)		
palpitations	6(14.29%)	7(12.07%)	1(3.85%)		

There was no significant difference in the distribution of chief complaints among different severity groups of coronary artery disease (chi-square = 2.75, p = 0.098). The percentages ofparticipants reporting breathlessness, chest pain, and chest pain with breathlessness, and palpitations were similara cross the SVD, DVD, and TVD groups.

Table-6: Distribution according to past history (comorbidities) among all groups

Past history (comorbidities)	SVD	DVD	TVD	Chi-square	P-value
	No of cases (%)	No of cases (%)	No of cases (%)		
Diabetes	12(28.57%)	13(22.41%)	7(26.92%)	6.90	0.008
Hypertension	3(7.14%)	11(18.97%)	7(26.92%)		
Hypertension And diabetes	1(2.38%)	3(5.17%)	0(0.00%)		
None	26(61.90%)	31(53.45%)	12(46.15%)		

The distribution of past medical history (comorbidities) differed significantly among severity groups of coronary artery disease (chi-square=6.90,p=0.008). Diabetes prevalence was highest in the SVD group (28.57%), followed by TVD (26.92%) and DVD (22.41%).Hypertension prevalence was highest in TVD(

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26.92%), followed by DVD (18.97%) and SVD (7.14%). None of the TVD group had both hypertension and diabetes, while small percentages in SVD (2.38%) andDVD (5.17%) did.

Table-7:Distributionaccordingtotobaccouse amongallgroups

Tobaccouse	SVD	DVD	TVD	Chi-square	P-value
	Noof cases (%)	Noof cases (%)	Noof cases (%)		
No	28(66.67%)	43(74.14%)	15(57.69%)	2.31	0.13
Yes	14(33.33%)	15(25.86%)	11(42.31%)		

Among participants, the percentages of non-users were as follows: SVD (66.67%), DVD(74.14%), and TVD (57.69%). Conversely, the percentages of tobacco users were: SVD (33.33%), DVD (25.86%), and TVD (42.31%). The distribution of tobacco use among severity groups of coronary artery disease did not show a significant difference (chi-square=2.31, p =0.13).

Table-8: Distribution according to Alcohol consumption among all groups

Alcohol consumption	SVD	DVD	TVD	Chi-square	P-value
	No of cases (%)	No of cases (%)	No of cases (%)		
No	25(59.52%)	39(67.24%)	13(50.00%)	2.31	0.12
Yes	17(40.48%)	19(32.76%)	13(50.00%)		

The majority of participants in each group reported not consuming alcohol: 59.52% in SVD, 67.24% in DVD, and 50.00% in TVD. Those who reported alcohol consumption representedsmaller percentages: 40.48% in SVD, 32.76% in DVD, and 50.00% in TVD.Alcohol consumption did not vary significantly across the severity groups studied. (Chi-square =2.31, p=0.12).

Table-9:Severity of coronary artery disease among participants

Severity of coronary artery disease	Noofcases	Percentage
DVD	58	46.04%
SVD	42	33.33%
TVD	26	20.63%
Total	126	100.00%

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Among total cases,58 cases (46.04%) had double vessel disease (DVD), 42 cases (33.33%) had single vessel disease (SVD),and 26 cases (20.63%) had triple vessel disease(TVD).

Table-10: Distribution according to lipid profile among all groups

Lipid Profile	SVD	DVD	TVD	f-test	P-value
	Mean±SD	Mean±SD	Mean±SD		
TotalCholesterol	197.81 ± 36.01	215.57 ± 31.13	215.57 ± 49.23	3.70	0.02
VLDL	34.57 ± 11.16	34.64 ± 7.63	39.31 ± 14.00	2.11	0.13
HDL(mg/dl)	41.14 ± 13.91	38.64 ± 8.15	37.5 ± 11.94	13.01	0.37
LDL(mg/dl)	146.83 ± 31.64	159.61 ± 27.92	165.8 ± 40.58	3.27	0.04
Triglyceride(mg/dl)	163.40 ± 48.49	181.89 ± 32.19	185.5 ± 38.19	3.52	0.03
APOA1(mg/dl)	90.67 ± 38.86	97.84 ± 39.42	83.78 ± 35.21	1.13	0.82
APOB(mg/dl)	125.68 ± 39.30	148.59 ± 39.34	148.59 ± 54.30	4.01	0.02
LDL/HDLRatio	4.08 ± 1.96	4.49 ± 2.17	5.40 ± 4.26	1.98	0.14
APOB/APOA1Ratio	1.65 ± 0.81	1.79 ± 0.81	2.23 ± 1.28	3.28	0.04

Total cholesterol levels were higher in DVD (215.57±31.13mg/dl) and TVD (215.57±49.23 mg/dl) compared to SVD (197.81 ± 36.01 mg/dl) (p = 0.02). LDL cholesterol wasalso higher in DVD (159.61 ± 27.92 mg/dl) and TVD (165.8 ± 40.58 mg/dl) compared toSVD (146.83 ± 31.64 mg/dl) (p = 0.04). Triglyceride levels were significantly different among groups, with higher values in DVD (181.89 ± 32.19 mg/dl) and TVD (185.5 ± 38.19mg/dl) compared to SVD (163.40 ± 48.49 mg/dl) (p = 0.03). APO B levels were elevated in DVD (148.59±39.34mg/dl) and TVD (148.59±54.30mg/dl) relative to SVD(125.68±39.30 mg/dl) (p = 0.02). Conversely, HDL levels, APO A1, LDL/HDL ratio did not showing insignificant differences across groups.

Table11: Comparison of age group with severity of CAD.

Agegroup	DVD	SVD	TVD	Total	Pvalue
<40years	9(27.27%)	21(63.64%)	3(9.09%)	33(100%)	0.009
>40years	49(52.69%)	21(22.58%)	23(24.73%)	93(100%)	

In patients under 40 years of age, 9 (27.27%) had DVD, 21 (63.64%) had SVD, and 3(9.09%) had TVD. Incontrast, among patients over 40 years of age, 49(52.69%) had DVD, 21(22.58%) had SVD, and 23(24.73%) had TVD. The p-value of 0.009 indicates sastatistically significant difference in the distribution of these vascular diseases between thetwo age groups. This data suggests that younger patients (<40 years) are more likely to have SVD, where as older patients (>40years) are more likely to have DVD and TVD.

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Table12: Distribution according lipid profile among all groups in less than 40 years.

Lipid Profile	SVD	DVD	TVD	f-test	P-value
	Mean± SD	Mean± SD	Mean± SD		
TotalCholesterol	200.93 ±39.83	201.428 ±39.83	252.33 ±37.02	5.36	0.15
VLDL	42 ± 10.21	38 ± 10.97	32.66 ±1.15	8.75	0.08
HDL(mg/dl)	37.53 ± 8.98	37.42 ± 4.75	36.33 ± 6.65	4.14	0.52
LDL(mg/dl)	163.1 ± 27.95	164 ± 33.50	189.15 ± 28.16	2.32	0.02
Triglyceride(mg/dl)	188.68 ± 39.18	172.5 ± 35.40	199.60 27.61	0.62	0.25
APOA1 (mg/dl)	78.88 ± 34.91	86.33 ± 15.01	73 ± 6.55	6.42	0.21
APOB(mg/dl)	136.27 ± 24.98	167.53 ± 26.99	209.83 ± 26.58	6.08	0.01
LDL/HDLRatio	4.74 ± 1.97	4.48 ± 1.33	5.24 ± 0.70	7.13	0.22
APOB/APOA1Ratio	1.95 ± 0.76	1.96 ± 0.31	2.90 ± 0.54	7.63	0.11

The table presents the distribution of lipid profiles among three groups (DVD, SVD, andTVD) for individuals under 40 years old, analyzing the data with an f-test and corresponding P-values. The mean total cholesterol levels were 201.428 ± 39.83 mg/dl for the DVD group, 200.93±39.83 mg/dl for the SVD group, and significantly higher at252.33± 37.02 mg/dl for the TVD group, showing a trend towards statistical significance (f-test:5.36, P-value: 0.15). VLDL levels varied, with 38 ± 10.97 mg/dl for DVD, 42 ± 10.21 mg/dlfor SVD, and 32.66 ± 1.15 mg/dl for TVD (f-test: 8.75, P-value: 0.08), indicating a potentialdifference. HDL levels were consistent across groups, with 37.42±4.75mg/dl (DVD), 37.53±8.98mg/dl (SVD), and 36.33±6.65mg/dl (TVD), showing no significant difference (f-test: 4.14, P-value: 0.52).LDL levels were 164±33.50 mg/dl (DVD), 163.1±27.95mg/dl (SVD), and higher at 189.15 ± 28.16 mg/dl (TVD), with a statistically significant difference (f-test: 2.32, P-value:0.02). Triglyceride levels were 172.5 ± 35.40 mg/dl (DVD), 188.68 ± 39.18 mg/dl (SVD),and 199.60±27.61mg/dl (TVD), with no significant differences (f-test:0.62, P-value 0.25). APO A1 levels were 86.33± 15.01 mg/dl (DVD), 78.88± 34.91mg/dl

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(SVD), and 73±6.55mg/dl (TVD), alsos howing no significant differences (f-test:6.42,P-value:0.21).

APO B levels were significantly different, with 167.53 ± 26.99 mg/dl (DVD), 136.27 ±24.98mg/dl (SVD), and 209.83±26.58mg/dl (TVD) (f-test:6.08,P-value:0.01).The LDL/HDL ratios were 4.48±1.33 (DVD), 4.74±1.97 (SVD), and 5.24±0.70 (TVD), Showing no significant differences (f-test: 7.13, P-value: 0.22). Finally, the APOB/APOA1 ratios were 1.96 ± 0.31 (DVD), 1.95 ± 0.76 (SVD), and higher at 2.90 ± 0.54 (TVD), indicating a potential difference but not statistically significant (f-test: 7.63, P-value: 0.11).Overall, significant differences were particularly noted in LDL and APO B levels among the groups, with theTVD group showing higher values.

Table13-Distribution according lipid profile among all groups in more than 40 years.

Lipid Profile	SVD	DVD	TVD	f-test	P-value
	Mean±SD	Mean±SD	Mean±SD		
Total Cholesterol	196.074 ± 34.38	217.509 ± 29.68	238.347 ± 51.25	2.96	0.22
VLDL	30.44 ± 9.52	34.17 ± 7.09	40.17 ± 14.69	6.83	0.18
HDL(mg/dl)	43.14 ± 15.18	38.80 ± 8.52	37.65 ± 12.56	4.46	0.24
LDL(mg/dl)	137.45 ± 30.38	159 ± 27.40	162.7 ± 41.46	7.74	0.02
Triglyceride(mg/dl)	149.25 ± 47.95	183.17 ± 31.89	183.65 ± 40.26	0.28	0.21
APOA1 (mg/dl)	91.21 ± 39.25	99.41 ± 41.10	85.19 ± 36.43	9.24	0.32
APOB(mg/dl)	119.79 ± 44.70	145.95 ± 40.22	147.81 ± 53.12	4.71	0.09
LDL/HDLRatio	3.71 ± 1.89	4.49 ± 2.27	5.42 ± 4.53	1.63	0.06
APOB/APOA1 Ratio	1.48 ± 0.79	1.76 ± 0.90	2.14 ± 1.32	4.49	0.15

The table presents the distribution of lipid profiles among three groups (DVD, SVD, and TVD) for individuals over 40 years old, including f-test values and P-values for statistical significance For Total Cholesterol, mean levels were 217.509 ± 29.68 mg/dl (DVD), 196.074 ± 34.38mg/dl (SVD), and 238.347 ± 51.25 mg/dl (TVD), with no significant difference (f-test: 2.96,P-value:0.22). VLDL levels were 34.17±7.09mg/dl (DVD), 30.44±9.52mg/dl (SVD), and 40.17±14.69mg/dl (TVD) (f-test:6.83,P-value:0.18), indicating no significant difference. HDL levels were consistent across groups (f-test: 4.46, P-value:0.24). LDL levels were 159±27.40mg/dl (DVD), 137.45±30.38mg/dl (SVD), and162.7±41.46mg/dl (TVD), showing a significant difference(f-test:7.74,P-value:0.02).

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DISCUSSION

Coronary artery disease (CAD) is a leading cause of mortality worldwide, affecting bothdeveloped and developing countries. [11] The rise in CAD can be attributed to an increase invariousrisk factors, including central obesity, diabetes, hypertension, atherogenic dyslipidemia, smoking, and physical inactivity. Recently, researchers have started to includenew biomarkers, such as Apolipoprotein A1 (Apo A1) and Apolipoprotein B (Apo B), along side traditional risk factors, to better assess theriskof CAD.[12]

Furthermore, low levels of HDL cholesterol, elevated levels of very low-density lipoprotein (VLDL) cholesterol, and high levels of triglycerides have been identified as significant riskfactors for CAD, particularly in patients with type 2 diabetes. These findings highlight theimportance of comprehensive lipid profiling in the assessment and management of CAD risk.[13]

There by, this prospective observational study was conducted to assess the correlation of lipid subfraction in patients with CAD. The study findings show that the TC, LDC, TyG, and Apo b have a significant relation with the CAD.

In this study, we included a total of 126 patients with angiographically proven CAD. The mean age of the patients was 56.75 years (SD = 16.11 years). The cohort was predominantly male (72.22%), with females constituting 27.78% of the sample.

Our findings align with those of previous studies. A study by **Zhang et al., [14]** which included 520 patients, reported a mean age of 56.6 years (SD = 10.0 years) and a male predominance of 64.4%. Similarly, **Xu et al., [15]** studied 413 patients and found a mean age of 55.12 years (SD =11.34 years), also with a highe rproportion of male patients.

Regarding the severity of CAD, we observed that among our 126 patients, 46.04% haddouble vessel disease (DVD), 33.33% had single vessel disease (SVD), and 20.63% had triple vessel disease (TVD).

Our study further revealed significant differences in mean age across the different severity groups: the mean age for patients with SVD was 46.21 years (SD±13.90), for DVDit was 60.52 years (SD ± 14.71), and for TVD it was 65.35 years (SD ± 13.82). The differences inage across these groups were statistically significant ($p<0.0001$), indicating that older age is associated with greater disease severity.

This observation is consistent with the findings of **Penalva et al., [16]** and supports thehypothesis that age is a significant factor in the progression and severity of CAD. These results which included 107 patients, of whom 94 (88%) had CAD.In their cohort, males with predominance of multivessel disease constituted 53.2%. They alsonoted that themean ages were higher in the two-vessel and multivessel groups compared to the one-vessel group (68±9 vs. 61±13 years, p=0.035). **Alshehri et al., [17]**al so show similarity to the present study reporting that the patients with multivessels disease were found to be older aged. In contrast, a study conducted by **Khashayar et al., [18]** and **Sharhan et al., [19]** found that age was not significantly affect the number of vessels occluded.

The present study found that height did not significantly differ across coronary artery disease (CAD) severity groups ($p = 0.81$), but weight ($p = 0.001$) and BMI ($p < 0.0001$) were significantly higher in participants with more severe disease, with those having triple-vessel disease (TVD) exhibiting the highest values (weight:76.92±16.55kg,BMI:28.68±7.39kg/m²).

Furthermore, there was no significant difference in the distribution of chief complaints (breathlessness, chest pain, ches pain with breathlessness, and ghabarahat/palpitation) among the different severity groups (chi-square = 2.75, $p = 0.098$), indicating that thenature of symptoms reported by participants was similar regardless of the severity of their CAD.

In the present study, the distribution of past medical history (comorbidities) significantly differed among severity groups of CAD in our study (chi square=6.90,p=0.008). Diabetes prevalence was highest among patients with single-vesseldisease (SVD) at 28.57%, followed by those with triple-vessel disease (TVD) at 26.92%, and double-vesseldisease (DVD) at 22.41%. Conversely, hypertension prevalence was highest in the TVD group at 26.92%, followed by DVD at 18.97%, and SVD at 7.14%. Notably, none of theTVD patients had both hypertension and diabetes, while small percentages of patients in the SVD(2.38%) and DVD (5.17%)groups wereobserved.

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These findings are consistent with a study by **Tanaka et al., [20]**which similarly reportedhigher hypertension prevalence in multi-vessel disease (96.8%) compared to two-vessel(81.5%) and one-vessel disease (78.1%). However, their study observed diabetes prevalenceashighest in multi-vesseldisease, contrastingwithourfindings.

In the present study, we observed that total cholesterol levels were higher in patient's withdoublevessel disease (DVD) (215.57±31.13 mg/dl). Triplevessel disease (TVD) (215.57 ±49.23 mg/dl) compared to those with single vessel disease (SVD) (197.81 ± 36.01 mg/dl), with a statistically significant difference (p = 0.02). Similarly, LDL cholesterol levels were elevated in the DVD (159.61 ± 27.92 mg/dl) and TVD (165.8 ± 40.58 mg/dl) groups relative to the SVD group (146.83 ± 31.64 mg/dl) (p = 0.04). Triglyceride levels also showed significant differences among the groups, being higher in DVDand TVD compared to SVD.

Apolipoprotein B (APO B) levels were elevated in the DVD (148.59 ± 39.34 mg/dl) and TVD (148.59±54.30mg/dl) groups compared to the SVD group (125.68±39.30mg/dl) (p

= 0.02). Similarly, HDL levels, Apolipoprotein A1 (APO A1) and LDL/HDL ratio did not show significant difference. The APOB/APOA1 ratio differ significantly across groups (p <0.05).

The result of this study was similar to another study conducted by **Tanaka et al., [20]**in which the LDL cholesterol levels were shown to be increased in the two-vessel group andmultivessel group. LDL levels had an average of 106.43 ± 40.51 mg/dl in the one-vessel group, 111.15 ± 39.43 mg/dl in the two-vessel group, and 114.52 ± 32.55 mg/dl in the multi-vessel group. There was no statistical significance found after the data was analyzed withone-way ANOVA (p =0.694).

These findings of the present study are also in line with the survey by **Penalva et al., [16]** which reported that LDL levels increased with the number of affected vessels. Patients withone-vessel disease had LDL levels of 85.54 ± 33.31 mg/dl, whereas those with multivessel disease had levels of 115.98±53.88mg/dl (p=0.077). Although the difference didnot reach statistical significance, likely due to the small sample size, the observation that patients with multivessel disease had LDL level sexceeding 100mg/dl is clinically significant. This is critical forthe prevention of CAD in high-risk patients.

Moreover, multiple studies have established a correlation between high levels of LDL andthe presence and severity of CAD. The study by **Korhonen et al.,** [21] further supports this,demonstrating that higher LDL levels are associated with an increased risk of CAD, with theseverity of obstruction correlating with serum LDL levels. This corroborates our findings, emphasizing the importanceofLDLmanagement inpatients with CAD.Furthermore, **Gruzdeva et al.,** [22] conducted a study demonstrating a notable associationbetween LDL levels and the severity of CAD in patients. Their approach involved strictadherence to angiography procedures and laboratory examinations conducted within a fewhours of disease onset, encompassing a substantial sample size of 400 individuals. These results show similarity with the present study.

In contrast, a study conducted by **Sharhan et al.,** [19] concluded that Triglyceride, TC/HDL,and LDL/HDLratios were not associated with the number of vessels affected.

Furthermore, research conducted by **Khashayar et al.**[18] found no correlation between these two variables. Their study indicated that besides LDL levels; high-density lipoprotein (HDL) is another factor influencing the extent of vessel disease.

Recent research emphasizes the diverse structural and functional characteristics of high-density lipoproteins (HDL), highlighting multiple dynamic subpopulations with varying degrees of atheroprotective properties. In a study conducted by **Sharhan et al., [19]**it waso bserved that LDL levels increased with the severity ofCAD, with patients diagnosed with multi-vessel disease showing elevated LDL levels (127.1 ± 45.7 mg/dl) compared to those with single-vessel disease(114 ± 34.3 mg/dl).

This finding corroborates earlier investigations by **Alsheri et al.,**[17] which found a significant correlation between LDL-C levels and the extent of CAD as determined by thenumber of affected vessels. Moreover, total cholesterol (TC), non-HDL cholesterol, and triglycerides (TG) were reported to be higher in patients

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JCHR (2026) 16(1), 1325-1336 | ISSN:2251-6727

with acute coronary syndrome (ACS) compared to those with stable angina, aligning with observations made by **Bhagwatet al.**[23]

In the present study, patients under 40 years exhibited a higher prevalence of Single Vessel Disease (SVD)(63.64%),where as those over 40 years had higherrates of Double Vessel Disease (DVD) (52.69%) and Triple Vessel Disease (TVD) (24.73%), with a statistically significantp-value of 0.009.

Similarly, **Faisal et al., [24]**findings shows that younger patients (≤ 35 years) shows higher occurrence of normal coronaries or mild CAD (40.6%) and clots with or without CAD(11.1%), while older patients (> 35 years) demonstrated higher incidences of DVD (25.7%),TVD (35.2%), and LMS(Left Main Stem) disease (4.5%), all with statistically significant p-values(<0.001forDVDand TVD, 0.002 for LMS).

Conclusion:

These findings highlight the clinical importance of examining lipoprotein profiles and the patterns of vessel involvement in coronaryartery disease (CAD), indicating potential implications for risk assessment and targeted the rapies. Further investigation in to the distinct roles played by various HDL subpopulations and their influence on atherosclerosis could advanceour knowledge and improve the management of cardiovascular conditions.

Additionally, our study adds evidence that elevated total cholesterol, LDL, and triglyceride levels are associated with more severe forms of CAD. The lack of significant differences inHDL,APOA1 levels and LDL/HDL ratio across the disease severity spectrum highlights the need for a comprehensivelipid profile, including LDL,triglycerides, APO A, APOB and its ratio in assessing and managing CAD risk. Future research should focus on larger sample sizes and the inclusion of additional biomarkers to furthere lucidates these relationships and improves clinical outcomes.

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