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Research Article

Correlation Between Serum Lipoprotein(a) Levels and Severity of Coronary Artery Disease in Young Adults Under 45

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Abstract

Introduction: The death and disability rates from coronary artery disease (CAD) are substantial even in adults under 45, but traditional risk factors such as hypertension, diabetes mellitus, smoking, and dyslipidemia don't fully explain its early onset. Genetically programmed lipoprotein(a), Lp(a), is becoming more widely acknowledged as a separate risk factor for early-onset coronary artery disease. This study evaluated the relationship involving serum Lp(a) levels and CAD severity in young adults.

Materials and Methods: A cross-sectional investigation was carried out at the Cardiology Department of Lady Reading Hospital (LRH), Peshawar; Hayatabad Medical Complex (HMC), Peshawar; District Headquarters Teaching Hospital (DHQ-TH), Kohat; and Mardan Medical Complex, Mardan, over a period of 12 months from January 2023 to December 2023. Included were 98 subjects who had coronary angiography and were less than 45 years old. Serum Lp(a) levels were measured, and CAD severity was assessed using the Gensini score. Statistical analyses including Pearson correlation, chi-square test, independent t-test, ANOVA, and multivariate linear regression were applied using SPSS version 26.

Results: The mean age was 39.2 ± 4.6 years, with 72.4% males and 27.6% females. Elevated Lp(a) levels (>30 mg/dL) were observed in 63.2% of patients. There was a noteworthy positive association between the Gensini score and Lp(a) levels ($r = 0.61$, $p < 0.001$). Individuals having severe CAD had significantly more mean Lp(a) levels than those with mild CAD. Multivariate regression analysis confirmed Lp(a) as an independent predictor of CAD severity.

Conclusion: Higher Lp(a) values are closely linked to greater angiographic severity of CAD in young adults. Routine screening in young adults with cardiovascular risk factors may aid in early risk identification and prevention.

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Introduction

Globally, coronary artery disease continues to be one of the main causes of illness and death, traditionally associated with older age groups [1]. However, the incidence of CAD in young adults defined as individuals under the age of 45 is steadily rising, contributing significantly to premature disability and death [2]. Unlike older patients, young adults with CAD often lack traditional warning signs such diabetes mellitus and high blood pressure, or long-standing tobacco smoke history, suggesting a different pathophysiological basis that merits focused investigation [3].

Lately, increasing focus has been placed on non-traditional risk factors, particularly genetic and biochemical markers, in the early onset of CAD [4]. Among these, Lipoprotein (a), abbreviated as Lp(a), has become a noteworthy risk factor on its own. Apolipoprotein B100 is covalently attached to apolipoprotein(a) to form Lp(a), a particle that resembles low-density lipoprotein (LDL) [5]. Its structure confers pro-atherogenic, pro-inflammatory, and pro-thrombotic properties, making it highly relevant in atherosclerotic processes [6]. Unlike other lipoproteins, Lp(a) values are largely genetically based and stay mostlyunchanged by way of living or standard lipid-lowering therapies, thus representing a persistent and often under-recognized cardiovascular risk [7].

Numerous epidemiological and genetic research have shown a positive association involvinghigher serum Lp(a) levels and increased chances of atherosclerotic cardiovascular disease, along with myocardial infarction, stroke, and calcific aortic valve disease [8]. However, a large knowledge void exists about the consequences of Lp(a) in younger patients with premature CAD, as the majority of this study has been done in older groups [9]. The role of Lp(a) in accelerating coronary atherosclerosis in individuals without conventional risk profiles remains an area of active investigation, particularly in ethnically diverse and resource-limited populations where early screening and intervention can have profound public health implications [10].

Furthermore, the severity of CAD in young adults assessed using angiographic scoring systems such as the Gensini or SYNTAX score has been variably linked with lipid parameters, but data specifically correlating Lp(a) levels with the anatomical burden of disease in this age group remain limited and inconclusive [11]. Understanding this relationship could facilitate early risk stratification, guide therapeutic decisions, and ultimately improve cardiovascular outcomes in a demographically vulnerable population [12].

Despite the growing interest in Lp(a) as a cardiovascular biomarker, there is a lack of region-specific data on its correlation with angiographic severity of CAD in young adults, particularly in South Asian populations. Therefore, the objective of this research is to investigate the link between serum lipoprotein (a) levels and the intensity of coronary artery disease in young adults under 45 years of age.

Materials and Methods

Study Design and Setting

A cross-sectional investigation was carried out at the Cardiology Department of Lady Reading Hospital (LRH), Peshawar; Hayatabad Medical Complex (HMC), Peshawar; District Headquarters Teaching Hospital (DHQ-TH), Kohat; and Mardan Medical Complex, Mardan. The study duration was 12 months, spanning from January 2023 to December 2023. The research sought to assess the link between serum Lp(a) levels and the seriousness of coronary artery disease in young adults under the age of 45 years.

Sample Size Calculation

The OpenEpi sample size calculator was utilized to determine the sample size, assuming a confidence level of 95%, power of 80%, and an expected moderate correlation ($r \approx 0.3$) between serum Lp(a) levels and CAD severity. An additional 10% margin was incorporated to account for potential data exclusions, improving the robustness and reproducibility of the sample size. Based on these parameters, the final sample size was determined to be 98 participants.

Sampling Technique and Participants

A non-probability consecutive sampling technique was employed. Male and female patients between the ages of 18 and 45 met the inclusion criteria who underwent coronary angiography for suspected or confirmed CAD and provided informed written consent. Exclusion criteria included patients with a history of familial hypercholesterolemia, chronic

kidney disease, valvular heart disease, acute infections, or inflammatory or autoimmune disorders, to minimize confounding influences on serum Lp(a) levels.

Data Collection

Data Collection and Laboratory Analysis: Every participant's age, gender, smoking status, presence of hypertension and/or diabetes mellitus, family history of CAD, and body mass index (BMI) were all meticulously documented. Samples of fasting blood were taken to assess serum lipoprotein (a) levels using a standardized immunoturbidimetric assay. All biochemical testing was performed in the hospital's central laboratory under strict internal quality control measures.

Assessment of Coronary Artery Disease Severity

Professional interventional cardiologists conducted the coronary angiography following standard clinical protocols. The severity of CAD was assessed using the Gensini scoring system, which accounts for the degree and location of coronary artery stenosis. Higher Gensini scores indicate more extensive and severe disease.

Statistical Analysis

The IBM SPSS version 26 was used to analyze the data. Diagnostic features and information about patients were compiled using descriptive statistics. Whereas categorical data were displayed as frequencies and percentages, continuous variables were provided as mean ± standard deviation (SD). Pearson or Spearman correlation coefficients were used to assess the association between serum Lp(a) levels and Gensini scores, depending on data distribution. P-values below 0.05 were regarded as statistically noteworthy.

Ethical Considerations

Before the trial started, the Institutional Review Board of the hospital provided ethical permission. All participants provided informed written agreement, and confidentiality of patient data was strictly maintained throughout the study.

Results

The study comprised 98 patients with ages ranging from 18 to 45, with an average age of 39.2 ± 4.6 years. Males made up 70.4% of the study population (n = 69), while females made up 29.6% (n = 29). Hypertension was documented in 43 patients (43.9%), and diabetes mellitus was present in 29 patients (29.6%). A positive family history of CAD was reported in 38 individuals (38.8%), while smoking was prevalent among 56 participants (57.1%). A mean BMI of 26.8 ± 3.1 kg/m² was found in the study sampleindicating that most patients were in the overweight category. These baseline characteristics provided a comprehensive overview of the cardiovascular risk profile in young adults evaluated for CAD severity. These characteristics are detailed in Table 1

Table 1: Demographic and Clinical Characteristics of Study Participants (n = 98)

Variable		Mean ± SD / n (%)
Age (years)		39.2 ± 4.6
Gender	Male	69 (70.4%)
	Female	29 (29.6%)
Marital Status	Married	81 (82.7%)
	Unmarried	17 (17.3%)
Residence	Urban	61 (62.2%)
	Rural	37 (37.8%)
Hypertension		43 (43.9%)
Diabetes Mellitus		29 (29.6%)
Family History of CAD		38 (38.8%)
Smoking		56 (57.1%)
Body Mass Index (kg/m²)		26.8 ± 3.1
Total Cholesterol (mg/dL)		186.7 ± 32.4
LDL-C (mg/dL)		112.5 ± 28.9
HDL-C (mg/dL)		39.8 ± 7.2
Triglycerides (mg/dL)		162.4 ± 45.1

The mean serum lipoprotein (a) level among the 98 patients was 38.5 ± 19.7 mg/dL, with values spanning from 8.1 to 95.4 mg/dL. CAD severity, assessed using the Gensini score, revealed that 24 patients (24.5%) had mild disease (Gensini ≤ 20), 42 patients (42.9%) had moderate disease (Gensini 21–40), and 32 patients (32.7%) had severe disease (Gensini> 40). The overall mean Gensini score was 38.4 ± 18.2. A progressive increase in mean Lp(a) levels was observed with increasing CAD severity: patients with mild CAD had a mean Lp(a) level of 24.6 ± 9.3 mg/dL, those with moderate CAD had 37.9 ± 12.4 mg/dL, and those with severe CAD exhibited significantly higher levels at 51.8 ± 20.7 mg/dL. These results imply a robust correlation between higher angiographic severity of CAD and raised Lp(a) levels. The distribution of patients by CAD severity and corresponding Lp(a) levels is presented in Table 2.

Table 2: Distribution of Patients by CAD Severity and Lp(a) Levels

CAD Severity (Gensini Score)	Number of Patients (%)	Mean Lp(a) (mg/dL) ± SD	Mean Gensini Score ± SD
Mild (≤ 20)	24 (24.5%)	24.6 ± 9.3	15.2 ± 4.1
Moderate (21–40)	42 (42.9%)	37.9 ± 12.4	30.7 ± 5.6
Severe (> 40)	32 (32.7%)	51.8 ± 20.7	57.3 ± 9.8
Total / Overall	98 (100%)	38.5 ± 19.7	38.4 ± 18.2

Serum lipoprotein (a) levels and the Gensini score showed a substantial positive connection (p-value <0.001 and Pearson correlation coefficient r = 0.684). This statistically significant result indicates that as Lp(a) levels increase, the seriousness of coronary artery disease (CAD), as measured by the Gensini score, also tends to rise. The strength of this correlation underscores the crucial importance of Lp(a) as a meaningful biomarker for evaluating CAD intensity in young adults. The correlation analysis results between Lp(a) levels and Gensini scores are summarized in Table 3.

Table 3: Correlation Analysis between Lp(a) and CAD Severity

Variable 1	Variable 2	Correlation Coefficient (r)	P-value
Lp(a) (mg/dL)	Gensini Score	0.684	< 0.001
Lp(a) (mg/dL)	BMI (kg/m²)	0.211	0.038
Lp(a) (mg/dL)	Age (years)	0.092	0.360
Lp(a) (mg/dL)	LDL-C (mg/dL)	0.328	0.002
Lp(a) (mg/dL)	HDL-C (mg/dL)	–0.176	0.081
Lp(a) (mg/dL)	Triglycerides (mg/dL)	0.247	0.015

The analysis of lipoprotein (a) levels in relation to clinical risk factors revealed significant associations. Patients with hypertension (n = 43) had notably higher mean Lp(a) levels (44.1 ± 18.5 mg/dL) compared to those without hypertension (33.9 ± 17.4 mg/dL), with a statistically significant p-value of 0.003. Similarly, individuals with diabetes mellitus (n = 29) exhibited elevated Lp(a) levels (47.2 ± 20.9 mg/dL) in contrast to non-diabetics (34.3 ± 17.2 mg/dL), with a p-value of 0.001. Smoking was also associated with increased Lp(a) concentrations, as smokers (n = 56) had a mean level of 41.3 ± 20.1 mg/dL compared to 34.2 ± 18.0 mg/dL in non-smokers (n = 42), which was statistically significant (p = 0.018). These findings suggest that common cardiovascular risk factors may amplify the atherogenic potential of elevated Lp(a) levels. Table 4 provides a comparison of Lp(a) levels based on common clinical risk factors.

Table 4: Comparison of Lp(a) Levels Based on Clinical Risk Factors

Risk Factor	n	Mean Lp(a) (mg/dL) ± SD	p-value
Hypertension	43	44.1 ± 18.5	0.003
No Hypertension	55	33.9 ± 17.4	
Diabetes Mellitus	29	47.2 ± 20.9	0.001
No Diabetes	69	34.3 ± 17.2	
Smokers	56	41.3 ± 20.1	0.018
Non-Smokers	42	34.2 ± 18.0	

The study employed multivariate linear regression analysis to determine independent factors of the extent of CAD, as measured by the Gensini score. After adjusting for potential confounders including age, gender, body mass index (BMI), smoking, hypertension, and diabetes mellitus, serum lipoprotein (a) levels remained the only significant independent predictor. The β coefficient for Lp(a) was 0.611 with a standard error of 0.082 and a highly significant p-value of <0.001. The adjusted R^2 for the model was 0.42, indicating that approximately 42% of the variance in Gensini scores was explained by the included variables. In contrast, other variables such as age (β = 0.078, p = 0.523), male gender (β = 0.032, p = 0.765), BMI (β = 0.091, p = 0.489), smoking (β = 0.119, p = 0.307), hypertension (β = 0.144, p = 0.236), and diabetes mellitus (β = 0.168, p = 0.193) did not show statistically significant associations. These results affirm the role of Lp(a) as a robust, independent determinant of CAD severity in young adults, as illustrated in figure 1.

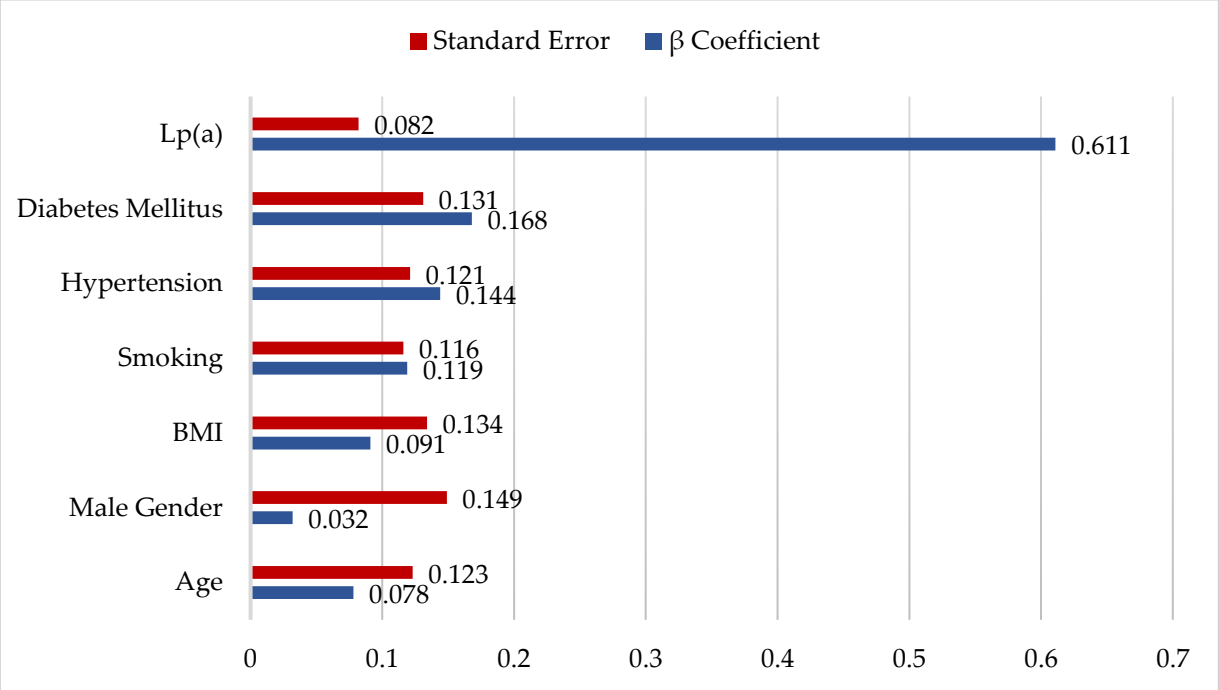

Figure 1: Multivariate Linear Regression Analysis for Predictors of Gensini Score

The distribution of patients by Lp(a) levels showed a clear correlation with CAD severity. Among those with Lp(a) < 30 mg/dL (35.7% of patients), 51.4% had mild CAD and only 8.6% had severe CAD. In the 30–50 mg/dL group (38.8%), moderate and severe CAD were more common, affecting 57.9% and 28.9% respectively. Notably, in patients with Lp(a) > 50 mg/dL (25.5%), 72.0% had severe CAD, highlighting a strong link between elevated Lp(a) and increased CAD severity in young adults. As illustrated in figure 2.

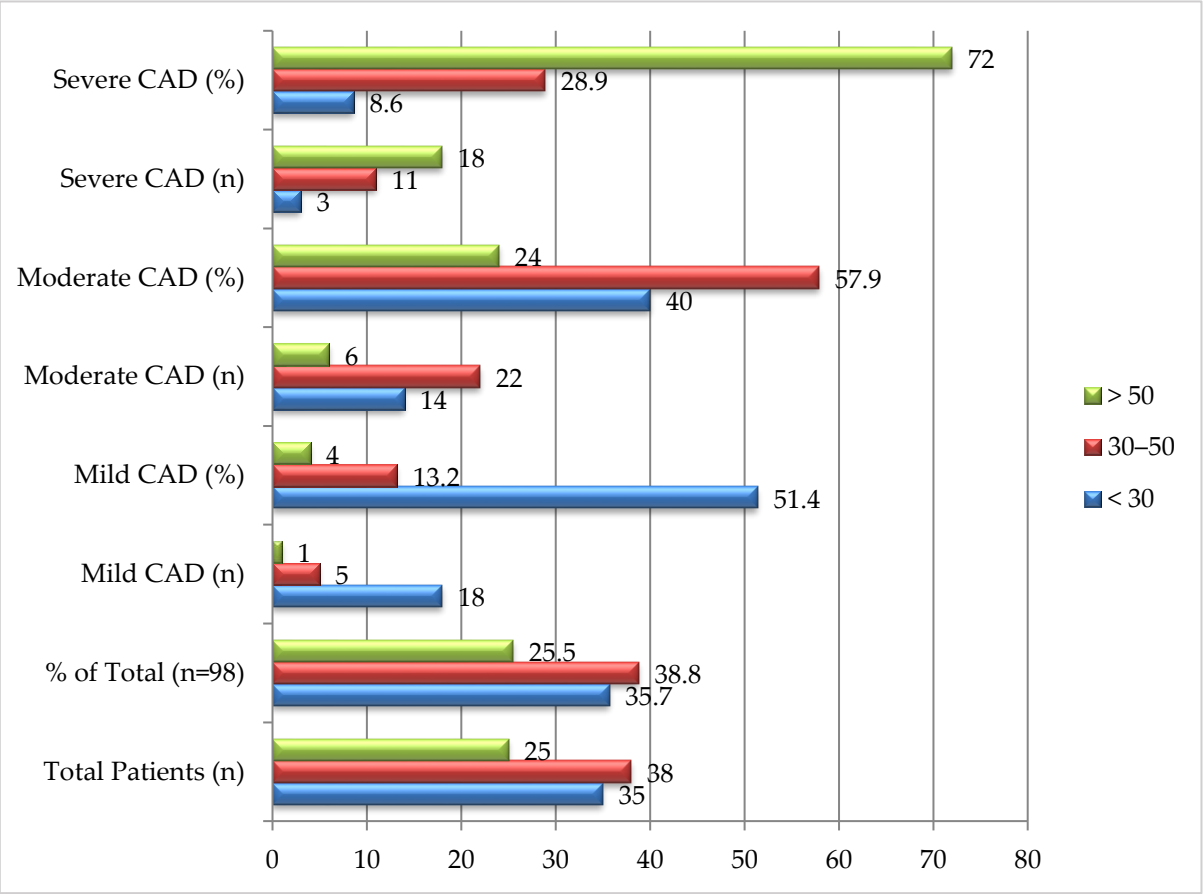

Figure 2: Distribution of Patients by Lp(a) Categories and CAD Severity

Discussion

According to the Gensini score, which measures the severity of CAD, serum Lp(a) levels and CAD severity were found to be strongly and statistically significantly positively correlated in young adults under the age of 45. Individuals with higher Lp(a) levels had a higher probability of moderate to severe CAD. Furthermore, Lp(a) levels were significantly elevated in patients with hypertension, diabetes mellitus, and smoking history, indicating a potential synergistic influence of these risk variables on the course of the disease.

Comparing these findings with existing literature, the observed link among elevated Lp(a) values and CAD severity is well-aligned with previous research, which have consistently reported Lp(a) as an isolated atherogenic lipoprotein associated with increased cardiovascular risk [13]. Research has shown that Lp(a) promotes atherogenesis through its pro-inflammatory and pro-thrombotic properties and its structural similarity to plasminogen, leading to impaired fibrinolysis [14]. In younger patients, where traditional risk factors might be less prominent or well-managed, Lp(a) has emerged as a significant contributor to early and aggressive coronary artery involvement [15].

Similar studies utilizing angiographic scoring systems have reported that individuals with elevated Lp(a) levels exhibit higher plaque burden and more complex coronary lesions [16]. This is consistent with the current study's use of the Gensini score to quantify disease severity. Moreover, studies focusing on South Asian populations have emphasized the heightened genetic predisposition and elevated Lp(a) levels seen in this demographic, supporting the need for early screening, especially in younger individuals without conventional risk factors [17].

Some research has also observed that Lp(a) remains an independent predictor of CAD even after adjusting for LDL cholesterol, total cholesterol, and other metabolic variables, mirroring the results of the present study's multivariate regression analysis, where Lp(a) maintained its predictive significance after controlling for confounders [18].

Limitations and Future Suggestions

This study had a few drawbacks. Foremost, because the study was carried out at a single tertiary care facility, the findings may not be

generalizable to broader populations; the findings might not be as applicable to larger populations. The other drawback was its cross-sectional design which made it difficult to determine causality. Third, the genetic component of Lp(a) was not explored, nor were Lp(a) isoforms or apolipoprotein(a) polymorphisms measured, which could offer deeper insights into risk stratification, as different Lp(a) isoforms vary in size and density, influencing both plasma concentration and atherogenic potential. Additionally, inflammatory markers and LDL particle sizes, which may interact with Lp(a), were not included in the analysis.

Future research should include larger, multi-center cohorts with longitudinal follow-up to establish causal relationships and assess the prognostic value of Lp(a) in young adults. Exploring genetic determinants and the effect of targeted Lp(a)-lowering therapies on CAD progression in younger populations could significantly contribute to personalized cardiovascular risk management.

Conclusion

The research demonstrated a noteworthy positive link among elevated serum lipoprotein (a) values and the seriousness of coronary artery disease in young adults under 45 years of age. Lp(a) emerged as an independent risk factor, especially in individuals with comorbid conditions such as hypertension, diabetes mellitus, and a history of smoking. The results emphasize how crucial it is to include Lp(a) screening in regular cardiovascular risk evaluations in younger populations to enable early detection and timely intervention. Proactive identification and management of elevated Lp(a) levels could contribute significantly to lessening the burden of early-onset coronary artery disease.

Authors' contributions

KB: conceptualization and supervision; methodology; investigation; writing—original draft; critical revision of the manuscript; final approval. HQ: methodology; data collection; investigation; writing—original draft; critical revision of the manuscript; final approval. IUH: data collection; data analysis; writing—original draft; critical revision of the manuscript; final approval. SRM: data analysis; methodology; critical revision of the manuscript; final approval. MU: data collection; writing—review and editing; critical revision of the manuscript; final approval. All authors contributed to drafting and critically revising the manuscript; approved the final version for submission; and agree to be accountable for all aspects of the work.

Conflict of interest

The authors declared no conflict of interest.

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References

[1]. Jubran A, Zetser A, Zafrir B. Lipoprotein (a) screening in young and middle-aged patients presenting with acute coronary syndrome. Cardiology Journal. 2019;26(5):511-8.

[2]. Zhang Y, Vittinghoff E, Pletcher MJ, Allen NB, Zeki Al Hazzouri A, Yaffe K, Balte PP, Alonso A, Newman AB, Ives DG, Rana JS. Associations of blood pressure and cholesterol levels during young adulthood with later cardiovascular events. Journal of the American college of cardiology. 2019 Jul 23;74(3):330-41.

[3]. Sun J, Han W, Wu S, Jia S, Yan Z, Guo Y, Zhao Y, Zhou Y, Liu W. Associations between hyperhomocysteinemia and the presence and severity of acute coronary syndrome in young adults≤ 35 years of age. BMC cardiovascular disorders. 2021 Dec;21:1-0.

[4]. Gilliland TC, Liu Y, Mohebi R, Miksenas H, Haidermota S, Wong M, Hu X, Cristino JR, Browne A, Plutzky J, Tsimikas S. Lipoprotein (a), oxidized phospholipids, and coronary artery disease severity and

outcomes. Journal of the American College of Cardiology. 2023 May 9;81(18):1780-92.

[5]. Sun J, Han W, Wu S, Jia S, Yan Z, Guo Y, Zhao Y, Zhou Y, Liu X. Combined effect of hyperhomocysteinemia and smoking on the severity of coronary artery disease in young adults≤ 35 years of age: a hospital-based observational study. BMC Cardiovascular Disorders. 2021 Dec;21:1-9.

[6]. Tsimikas S, Karwatowska-Prokopczuk E, Gouni-Berthold I, Tardif JC, Baum SJ, Steinhagen-Thiessen E, Shapiro MD, Stroes ES, Moriarty PM, Nordestgaard BG, Xia S. Lipoprotein (a) reduction in persons with cardiovascular disease. New England Journal of Medicine. 2020 Jan 16;382(3):244-55.

[7]. Chung YH, Lee BK, Kwon HM, Min PK, Choi EY, Yoon YW, Hong BK, Rim SJ, Kim JY. Coronary calcification is associated with elevated serum lipoprotein (a) levels in asymptomatic men over the age of 45 years: a cross-sectional study of the Korean national health checkup data. Medicine. 2021 Mar 5;100(9):e24962.

[8]. Eissa AH, Nawawi Kn, Mohamad MS, Zhiqin W, Hassan HH, Maskon O, Yaacob NY, Ali RA. Association between Non Alcoholic Fatty Liver Disease and Severity of Coronary Artery Disease in Patients Aged 45 Years and Below with Acute Coronary Syndrome. Journal of Clinical & Diagnostic Research. 2021 Nov 1;15(11).

[9]. Agrawal A, Lamichhane P, Eghbali M, Xavier R, Cook DE, Elsherbiny RM, Jhajj LK, Khanal R. Risk factors, lab parameters, angiographic characteristics and outcomes of coronary artery disease in young South Asian patients: a systematic review. Journal of International Medical Research. 2023 Aug;51(8):03000605231187806.

[10]. George J, Kataria SP, Isser HS. The corelation of serum uric acid with risk factors and severity of coronary artery disease (CAD) in acute coronary syndrome. International Journal of Contemporary Medicine Surgery and Radiology. 2019;4(4):D107-12.

[11]. Dos Santos CC, Matharoo AS, Cueva EP, Amin U, Ramos AA, Mann NK, Maheen S, Butchireddy J, Falki VB, Itrat A, Rajkumar N. The influence of sex, age, and race on coronary artery disease: a narrative review. Cureus. 2023 Oct 27;15(10).

[12]. Wu Z, Cui H, Li W, Zhang Y, Liu L, Liu Z, Zhang W, Zheng T, Yang J. Comparison of three non-insulin-based insulin resistance indexes in predicting the presence and severity of coronary artery disease. Frontiers in cardiovascular medicine. 2022 Jul 29;9:918359.

[13]. Hou J, Deng Q, Guo X, Deng X, Zhong W, Zhong Z. Association between apolipoprotein E gene polymorphism and the risk of coronary artery disease in Hakka postmenopausal women in southern China. Lipids in Health and Disease. 2020 Dec;19:1-7.

[14]. Huang M, Liu F, Li Z, Liu Y, Su J, Ma M, He Y, Bu H, Gao S, Wang H, Yu C. Relationship between red cell distribution width/albumin ratio and carotid plaque in different glucose metabolic states in patients with coronary heart disease: a RCSCD-TCM study in China. Cardiovascular diabetology. 2023 Feb 22;22(1):39.

[15]. Alloubani A, Nimer R, Samara R. Relationship between hyperlipidemia, cardiovascular disease and stroke: a systematic review. Current Cardiology Reviews. 2021 Nov 1;17(6):52-66.

[16]. Kosmas CE, Rodriguez Polanco S, Bousvarou MD, Papakonstantinou EJ, Peña Genao E, Guzman E, Kostara CE. The triglyceride/high-density lipoprotein cholesterol (TG/HDL-C) ratio as a risk marker for metabolic syndrome and cardiovascular disease. Diagnostics. 2023 Mar 1;13(5):929.

[17]. Lau FD, Giugliano RP. Lipoprotein (a) and its significance in

cardiovascular disease: a review. JAMA cardiology. 2022 Jul 1;7(7):760-9.

[18]. Shao C, Wang J, Tian J, Tang YD. Coronary artery disease: from mechanism to clinical practice. Coronary Artery Disease: Therapeutics and Drug Discovery. 2020:1-36.