

Association of Lipoprotein(a) With SYNTAX Score in Young Adults Presenting with Acute Coronary Syndrome: A Cross-Sectional Study

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Abstract

Background

Premature coronary artery disease is increasingly recognized as a major public health concern, particularly in developing countries. Lipoprotein(a) [Lp(a)] has emerged as an important genetically determined cardiovascular risk factor; however, its relationship with angiographic severity of coronary artery disease in young adults presenting with acute coronary syndrome (ACS) remains inadequately explored. This study aimed to evaluate the association between Lp(a) levels and coronary angiographic severity assessed by SYNTAX score in young adults with ACS.

Methods

This single-center cross-sectional study was conducted in the Department of Cardiology, AIIMS Rishikesh. Fifty-five consecutive patients aged ≤ 45 years presenting with ACS were enrolled. Clinical characteristics, cardiovascular risk factors, laboratory investigations, and coronary angiographic findings were recorded. Serum Lp(a) levels were measured using standard laboratory techniques. Coronary angiographic complexity was assessed using the SYNTAX score and categorized as low (0–22), intermediate (23–32), and high (>32). Associations between Lp(a) and SYNTAX score were analyzed using Chi-square and Spearman correlation analyses.

Results

The study population comprised predominantly males (85.5%), with a mean age of 39.8 ± 5.4 years. ST-elevation myocardial infarction was the most common presentation (76.4%). The mean Lp(a) level was 47.96 ± 29.35 mg/dL, and 47.3% of patients were categorized as high risk based on Lp(a) levels. The mean SYNTAX score was 15.34 ± 7.40 . A significant association was observed between Lp(a) categories and SYNTAX score severity ($\chi^2 = 19.638$, $p < 0.001$). Furthermore, Lp(a) demonstrated a moderate positive correlation with SYNTAX score (Spearman $r = 0.540$, $p < 0.001$), indicating greater angiographic complexity among patients with elevated Lp(a) levels.

Conclusions

Elevated Lp(a) levels are significantly associated with increased coronary angiographic severity in young adults presenting with ACS. Assessment of Lp(a) may help identify high-risk individuals with more complex coronary artery disease and could serve as a useful adjunct in risk stratification of premature ACS.

Background

Premature coronary artery disease (CAD) remains a major public health challenge, particularly in South Asian populations, where cardiovascular events frequently occur at a younger age than in Western countries [1, 2]. Acute coronary syndrome (ACS) in adults aged 45 years or younger is associated with

substantial morbidity, premature loss of productive life-years, and significant socioeconomic burden. Despite advances in preventive cardiology, the incidence of premature ACS continues to rise, underscoring the need for improved risk stratification and identification of novel cardiovascular risk factors [1, 3].

Although traditional risk factors such as smoking, diabetes mellitus, hypertension, obesity, and dyslipidemia contribute significantly to premature CAD, these factors alone do not fully explain the occurrence or severity of coronary disease in many young patients [3, 4]. Consequently, increasing attention has been directed toward genetically determined risk factors that may provide incremental information beyond conventional cardiovascular risk assessment.

Lipoprotein(a) [Lp(a)] has emerged as one of the most important inherited cardiovascular risk factors. Structurally, Lp(a) consists of an apolipoprotein B-containing low-density lipoprotein particle linked to apolipoprotein(a), a highly polymorphic glycoprotein [5]. Circulating Lp(a) concentrations are largely genetically determined and remain relatively unaffected by lifestyle modification or conventional lipid-lowering therapies [5, 6]. Contemporary evidence from genetic, epidemiological, and clinical studies has firmly established elevated Lp(a) as an independent and causal risk factor for atherosclerotic cardiovascular disease and calcific aortic valve disease [5–7].

The pathogenic effects of Lp(a) are mediated through multiple mechanisms. The LDL-like component promotes cholesterol accumulation within the arterial wall, while apolipoprotein(a) contributes to vascular inflammation, oxidative stress, endothelial dysfunction, and impaired fibrinolysis [5, 8]. These properties accelerate atherosclerotic plaque development and progression, increasing the risk of myocardial infarction and adverse cardiovascular outcomes. Recent international guidelines and consensus statements have therefore recommended at least one lifetime measurement of Lp(a) to improve cardiovascular risk assessment [5, 6].

Several contemporary studies have demonstrated a significant association between elevated Lp(a) concentrations and angiographic severity of CAD [9–12]. Patients with increased Lp(a) levels have been shown to exhibit a higher burden of coronary atherosclerosis, more complex coronary lesions, and greater prevalence of multivessel disease. Recent evidence has further suggested that elevated Lp(a) is associated with higher SYNTAX scores, indicating increased anatomical complexity of coronary artery disease [10–12].

The SYNTAX score is a validated angiographic tool that quantifies both the extent and complexity of coronary artery disease and plays an important role in prognostication and revascularization decision-making [13]. However, despite growing evidence supporting the clinical significance of Lp(a), data evaluating its relationship with angiographic severity in young Indian adults presenting with ACS remain limited. Given the disproportionately high burden of premature CAD in India and the increasing recognition of Lp(a) as a modifiable therapeutic target, further investigation in this population is warranted.

Therefore, the present study aimed to evaluate the association between serum Lipoprotein(a) levels and coronary angiographic severity assessed by the SYNTAX score in young adults (≤ 45 years) presenting with acute coronary syndrome at a tertiary care center in Northern India.

Methods

Study Design and Setting

This single-center cross-sectional study was conducted in the Department of Cardiology, All India Institute of Medical Sciences (AIIMS), Rishikesh, Uttarakhand, India. The study was carried out over a period of 18 months following approval from the Institutional Ethics Committee. All participants provided written informed consent before enrollment.

Study Population

Consecutive patients aged ≤ 45 years presenting with acute coronary syndrome (ACS) and admitted to the Department of Cardiology were screened for inclusion. ACS included ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina (UA), diagnosed according to contemporary guideline-based clinical, electrocardiographic, and biochemical criteria.

As this was a time-bound exploratory study, a convenience sample was employed. A total of 55 patients fulfilling the eligibility criteria were included in the final analysis.

Inclusion and Exclusion Criteria

Patients aged ≤ 45 years with a diagnosis of ACS who underwent coronary angiography were eligible for inclusion.

Patients were excluded if they had a prior history of ACS, percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), chronic kidney disease, cardiomyopathy, significant valvular heart disease, pericardial disease, malignancy, pregnancy, known contrast allergy, refusal to undergo coronary angiography, or refusal to provide informed consent.

Clinical and Laboratory Assessment

Baseline demographic characteristics, cardiovascular risk factors, clinical presentation, and laboratory parameters were recorded at admission. Information regarding smoking status, diabetes mellitus, hypertension, obesity, and family history of premature coronary artery disease was obtained through detailed history taking and review of medical records.

All patients underwent 12-lead electrocardiography and transthoracic echocardiography. Left ventricular ejection fraction and regional wall motion abnormalities were assessed using standard echocardiographic techniques.

Measurement of Lipoprotein(a)

Venous blood samples were collected under standardized conditions during hospitalization. Serum Lipoprotein(a) [Lp(a)] levels were measured in the central laboratory using an immunoturbidimetric assay according to the manufacturer's protocol. Based on laboratory reference ranges, patients were categorized into low-risk, intermediate-risk, and high-risk groups for subsequent analysis.

Coronary Angiography and SYNTAX Score Assessment

All patients underwent coronary angiography using standard percutaneous techniques through either radial or femoral arterial access. Coronary angiograms were obtained in multiple orthogonal projections and interpreted by experienced cardiologists.

Significant coronary artery disease was defined as $\geq 70\%$ luminal stenosis in the left anterior descending artery, left circumflex artery, or right coronary artery, and $\geq 50\%$ stenosis in the left main coronary artery. Based on angiographic findings, patients were categorized as having single-vessel disease (SVD), double-vessel disease (DVD), or triple-vessel disease (TVD).

Results

Table 1
Demographic details of patients

Gender	N	%
Female	8	14.5%
Male	47	85.5%

Gender of study patients are displayed in Table 1. Males made up the majority of patients (85.5%), with only 14.5% of the study population being female.

Table 2
Distribution of Age among Young MI patients

Age Distribution	N	%
20–30 years	7	12.7%
31–40 years	20	36.4%
41–45 years	28	50.9%

With reference to Table 2, the age group of 41–45 years old made up the majority of participants (50.9%), followed by 31–40 years old (36.4%). Just 12.7% of participants were in the 20–30 age range. This suggests that middle-aged male participants in the current study had a higher prevalence of CAD.

Table 3
Distribution of Type of Acute
Coronary Syndrome among
patients

Type of MI	N	%
STEMI	42	76.4%
AWMI	28	50.9%
IWMI	14	25.5%
NSTEMI	9	16.4%
UA	4	7.3%

Table 3 demonstrates that, STEMI patients contribute 42(76.4%) with 50.9% of patients had anterior wall myocardial infarction (AWMI) followed by 25.5% had inferior wall myocardial infarction (IWMI). Of the patients, 16.4% had NSTEMI, and 7.3% had unstable angina (USA). These findings indicate that AWMI was the predominant clinical presentation among CAD patients.

Table 4
Coronary Angiographic
Findings among Patients

CAG	N	%
SVD	34	61.8%
DVD	9	16.4%
TVD	11	20.8%
LMCA + TVD	1	1.80%

Table 4 shows that, in 61.8% of patients, single vessel disease (SVD) was the most prevalent angiographic finding. 20.8% of patients had triple vascular disease (TVD), whereas 16.4% had double vessel disease (DVD). 1.80% of patients had TVD with LMCA.

Table 5
Distribution of Novel Biomarkers i.e Lipoprotein(a) in young
CAD (age ≤ 45)

Novel Biomarkers (Mean ± SD)		N	%
Lipoprotein(a) (47.96 ± 29.345)	Low Risk (< 30 mg/dl)	21	38.2%
	Intermediate Risk (30–50 mg/dl)	8	14.5%
	High Risk (> 50 mg/dl)	26	47.3%

The distribution of novel biomarkers of atherosclerosis among patients shown in Table 5. With a mean Lipoprotein(a) level of 47.96 ± 29.345 , over almost half of the patients (47.3%) were classified as high-risk, whereas 38.2% and 14.5% were classified as low-risk and intermediate-risk, respectively.

Table 6
Distribution of Syntax Score in young ACS patients

Syntax Score	Mean = 15.34, SD = 7.399	
	N	%
Low Syntax	39	70.9%
Intermediate	15	27.3%
High Syntax	1	1.8%

The distribution of SYNTAX score categories among study participants is shown in Table 6. The majority of patients (70.9%) had a low SYNTAX score, while 27.3% had an intermediate SYNTAX score and only 1.8% had a high SYNTAX score. The mean SYNTAX score of the study population was 15.34 ± 7.40

Table 7
Association between Lipoprotein(a) and SYNTAX Score

		SYNTAX						Chi Square (p value)
		Low Syntax		Intermediate		High Syntax		
		N	%	N	%	N	%	
Lipoprotein(a)	Low Risk	20	36.4%	1	1.8%	0	0.0%	19.638 (< 0.001)
	Intermediate Risk	8	14.5%	0	0.0%	0	0.0%	
	High Risk	11	20.0%	14	25.5%	1	1.8%	

Using the Chi-square test, Table 7 displays the relationship between lipoprotein(a) and SYNTAX score categories among research participants. Higher levels of lipoprotein(a) were linked to more severe coronary artery disease, according to a statistically significant correlation found between Lipoprotein(a) and SYNTAX score ($\chi^2 = 19.638$, $p < 0.001$).

Table 8
Correlation between serum Lipoprotein(a) levels and the severity of coronary artery disease, as assessed by the SYNTAX score, was evaluated using Spearman's correlation analysis

Spearman's Correlation Analysis		
	Correlation Coefficient	P value
Lipoprotein (a) Vs SYNTAX	0.540	< 0.001

Spearman's correlation analysis was utilized to evaluate the link between new biomarkers and the severity of coronary artery disease. The SYNTAX score and lipoprotein(a) had a moderately positive connection ($r = 0.540$, $p < 0.001$). This finding indicates that higher Lipoprotein(a) concentrations are associated with greater angiographic severity and complexity of coronary artery disease

Figure 1: illustrates the relationship between serum Lipoprotein(a) levels and SYNTAX score among young adults presenting with acute coronary syndrome

Fig. 1: Scatter plot demonstrating a moderate positive correlation between Lipoprotein(a) concentration and SYNTAX score (Spearman's correlation coefficient $r = 0.540$, $p < 0.001$). Higher Lipoprotein(a) levels were associated with increased angiographic complexity and severity of coronary artery disease

Discussion

In the present cross-sectional study of young adults (≤ 45 years) presenting with acute coronary syndrome, we observed a significant association between elevated serum Lipoprotein(a) [Lp(a)] levels and increased angiographic severity of coronary artery disease as assessed by the SYNTAX score. Nearly half of the study population (47.3%) belonged to the high-risk Lp(a) category, and elevated Lp(a) levels were associated with progressively higher SYNTAX score categories. Furthermore, Lp(a) demonstrated a moderate positive correlation with SYNTAX score ($r = 0.540$, $p < 0.001$), suggesting that higher Lp(a) concentrations are associated with more complex coronary artery disease. These findings support the growing body of evidence implicating Lp(a) as an important determinant of premature and severe coronary atherosclerosis [3, 5, 8].

Premature ACS is a distinct clinical entity characterized by a high prevalence of modifiable and non-modifiable cardiovascular risk factors. In our study, males constituted 85.5% of the study population, and the majority of patients belonged to the 41–45-year age group. These findings are consistent with previous studies from India and other South Asian countries demonstrating a marked male predominance among young patients presenting with myocardial infarction [1, 2]. The relatively younger age at presentation observed in Indian populations compared with Western cohorts highlights the aggressive nature of atherosclerotic cardiovascular disease in this region and emphasizes the need for improved risk stratification strategies [1, 2].

Among conventional cardiovascular risk factors, smoking was the most prevalent risk factor in our cohort, being present in 52.7% of patients, followed by diabetes mellitus and hypertension. These observations are in agreement with previous reports indicating that tobacco exposure remains one of the strongest contributors to premature coronary artery disease [2, 4]. However, conventional risk factors alone may not fully explain the occurrence and severity of CAD in young individuals. This has led to increasing interest in genetically mediated risk factors such as Lp(a), which may provide additional prognostic information beyond traditional cardiovascular risk assessment [3, 5].

Lp(a) has emerged as one of the most important inherited cardiovascular risk factors. Unlike conventional lipid parameters, plasma Lp(a) concentrations are predominantly genetically determined and remain relatively stable throughout life [3, 6]. Recent genetic, epidemiological, and mechanistic studies have established elevated Lp(a) as a causal risk factor for atherosclerotic cardiovascular disease [3, 5, 7]. Current international guidelines and expert consensus statements recommend at least one lifetime measurement of Lp(a), particularly in individuals with premature cardiovascular disease, a family history of early CAD, or unexplained cardiovascular risk [3, 5]. In our study, the mean Lp(a) concentration was 47.96 ± 29.35 mg/dL, and nearly half of the participants were categorized as high risk, emphasizing the substantial burden of elevated Lp(a) among young ACS patients.

The most important finding of the present study was the significant relationship between elevated Lp(a) levels and angiographic severity of coronary artery disease. Patients with higher Lp(a) concentrations were more likely to demonstrate intermediate or high SYNTAX scores, indicating greater anatomical complexity of coronary lesions. The observed moderate positive correlation between Lp(a) and SYNTAX score further strengthens this association. These findings are biologically plausible given the unique pathogenic characteristics of Lp(a) [3, 5, 8]. The LDL-like component facilitates cholesterol accumulation within the arterial wall, whereas apolipoprotein(a) promotes endothelial dysfunction, vascular inflammation, oxidative stress, and impaired fibrinolysis [3, 5, 8]. Together, these mechanisms accelerate plaque formation and progression, ultimately contributing to more diffuse and complex coronary artery disease.

Our findings are consistent with several previous studies evaluating the relationship between Lp(a) and angiographic severity of CAD. Ashfaq et al. demonstrated a significant association between elevated Lp(a) levels and higher SYNTAX scores among patients undergoing coronary angiography [14]. Similarly, recent studies have reported that elevated Lp(a) concentrations are associated with multivessel disease, greater atherosclerotic burden, and increased coronary lesion complexity [9–12]. Kozieł-Siołkowska and colleagues reported that elevated Lp(a) independently predicted higher SYNTAX scores [9], while other contemporary investigations have demonstrated similar associations across different ethnic populations [10–12]. The consistency of these observations across multiple studies suggests that Lp(a) is not merely a marker of cardiovascular risk but may actively contribute to the development of anatomically severe coronary artery disease.

An important clinical implication of our findings is the potential role of Lp(a) in risk stratification among young patients presenting with ACS. Traditional risk assessment tools frequently underestimate cardiovascular risk in younger individuals because of their heavy reliance on age-based algorithms [5]. Measurement of Lp(a) may help identify a subgroup of young patients with genetically mediated susceptibility to accelerated atherosclerosis and more severe coronary involvement [3, 5, 6]. Early recognition of elevated Lp(a) may facilitate implementation of aggressive preventive strategies, including intensive lipid-lowering therapy, optimization of modifiable cardiovascular risk factors, and closer long-term surveillance. Furthermore, the emergence of novel Lp(a)-lowering therapies currently undergoing clinical evaluation highlights the growing therapeutic relevance of identifying patients with elevated Lp(a) [7].

The strengths of our study include its focus on a relatively underexplored population of young adults with ACS, comprehensive angiographic assessment using the validated SYNTAX score [13], and evaluation of Lp(a), a clinically important and increasingly recognized cardiovascular risk factor. The study provides valuable data from an Indian tertiary-care center, where premature CAD continues to represent a major healthcare challenge.

However, several limitations should be acknowledged. First, this was a single-center study with a relatively modest sample size, which may limit the generalizability of the findings. Second, the cross-sectional design precludes assessment of causal relationships and long-term clinical outcomes. Third, Lp(a) levels were measured only once during hospitalization. Finally, residual confounding from unmeasured variables cannot be completely excluded. Larger multicenter prospective studies are required to validate these findings and determine the prognostic significance of elevated Lp(a) in young patients with ACS.

Conclusions

In this cross-sectional study of young adults (≤ 45 years) presenting with acute coronary syndrome, elevated Lipoprotein(a) [Lp(a)] levels were significantly associated with increased angiographic severity of coronary artery disease as assessed by the SYNTAX score. Patients with higher Lp(a) concentrations demonstrated greater coronary lesion complexity and a higher burden of atherosclerotic disease. Furthermore, Lp(a) showed a moderate positive correlation with SYNTAX score, supporting its potential role as a marker of severe coronary artery disease in young individuals.

Given its predominantly genetic determination and relative independence from conventional cardiovascular risk factors, Lp(a) may provide incremental value in identifying young patients at increased risk of complex coronary artery disease. Routine assessment of Lp(a) in young adults presenting with acute coronary syndrome may facilitate improved risk stratification and support more intensive preventive and therapeutic strategies. Larger multicenter prospective studies are warranted to further validate these findings and establish the prognostic and therapeutic implications of elevated Lp(a) in premature coronary artery disease.

Abbreviations

ACS

Acute Coronary Syndrome

AWMI

Anterior Wall Myocardial Infarction

CABG

Coronary Artery Bypass Grafting

CAD

Coronary Artery Disease

CAG

Coronary Angiography

DVD

Double Vessel Disease

ECG

Electrocardiography

HTN

Hypertension

LAD

Left Anterior Descending Artery

LCX

Left Circumflex Artery

LDL-C

Low-Density Lipoprotein Cholesterol

LMCA

Left Main Coronary Artery

Lp(a)

Lipoprotein(a)

LVEF

Left Ventricular Ejection Fraction

NSTEMI

Non-ST-Segment Elevation Myocardial Infarction

PCI

Percutaneous Coronary Intervention

RCA

Right Coronary Artery

RWMA

Regional Wall Motion Abnormality

STEMI

ST-Segment Elevation Myocardial Infarction

SVD
Single Vessel Disease
SYNTAX
Synergy Between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery
TVD
Triple Vessel Disease
UA
Unstable Angina

Declarations

Trial registration

Not applicable. This was a cross-sectional observational study

Ethics approval and consent to participate

The study was approved by the Institutional Ethics Committee of All India Institute of Medical Sciences (AIIMS), Rishikesh. Written informed consent was obtained from all participants prior to enrolment in the study.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Akash Swain conceived the study, collected data, performed data analysis, and drafted the manuscript. Barun Kumar supervised the study, contributed to study design, interpretation of results, and critically revised the manuscript. Kanika Kukreja, Aditya Agarwal, and Kishan Mavani contributed to data collection, data interpretation, and manuscript review. All authors read and approved the final manuscript.

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References

1. Di Cesare M, Perel P, Taylor S, Kabudula CW, Bixby H, Gaziano TA, et al. The heart of the world. *Glob Heart*. 2024;19(1):11.
2. Peerwani G, Karthikeyan G, Rao SV, Xavier D, Pais P, Sharma M, et al. Presentation, management, and early outcomes of young patients with acute coronary syndrome: insights from a contemporary registry. *Indian Heart J*. 2024;76:188–195.
3. Kronenberg F, Mora S, Stroes ESG, Ference BA, Arsenault BJ, Berglund L, et al. Lipoprotein(a) in atherosclerotic cardiovascular disease and aortic stenosis: a European Atherosclerosis Society consensus statement. *Eur Heart J*. 2022;43(39):3925–3946.
4. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. *Eur Heart J*. 2020;41(1):111–188.
5. Virani SS, Newby LK, Arnold SV, Bittner V, Brewer LC, Demeter SH, et al. 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA guideline for the management of patients with chronic coronary disease. *Circulation*. 2023;148:e9–e119.
6. Tsimikas S, Marcovina SM. Ancestry, lipoprotein(a), and cardiovascular risk thresholds: JACC review topic of the week. *J Am Coll Cardiol*. 2022;80(9):934–946.
7. O'Donoghue ML, Rosenson RS, Gencer B, López JAG, Lepor NE, Baum SJ, et al. Small interfering RNA to reduce lipoprotein(a) in cardiovascular disease. *N Engl J Med*. 2022;387(20):1855–1864.
8. Leistner DM, Laguna-Fernandez A, Scharnagl H, Zeiher AM, März W, Landmesser U, et al. Impact of elevated lipoprotein(a) on coronary artery disease phenotype and severity. *Eur J Prev Cardiol*. 2024;31(7):856–865.
9. Kozieł-Siołkowska M, Krawczyk-Ożóg A, Lelakowski J, Płońska-Gościński E, Nessler J, Stępień E, et al. Lipoprotein(a) as an independent predictor of elevated SYNTAX score. *J Clin Med*. 2024;13(23):7109.
10. Sandhu AS, Ahmed S, Singh S, Kaur M, Sharma A, et al. Serum lipoprotein(a) and angiographic severity of coronary artery disease in an Indian population. *Res Cardiovasc Med*. 2024;13(2):77–83.
11. Shui X, Wen Z, Chen Z, Xie X, Wu Y, Zheng B, et al. Elevated serum lipoprotein(a) is significantly associated with angiographic progression of coronary artery disease. *Clin Cardiol*. 2021;44(11):1551–1559.
12. Xu W, Guan H, Gao D, Wang Z, Ba Y, Yang H, et al. The association of SYNTAX score with levels of lipoprotein(a) and inflammatory biomarkers in patients with stable coronary artery disease and

different low-density lipoprotein cholesterol levels. *Diabetes Metab Syndr Obes.* 2020;13:4297–4310.

13. Sianos G, Morel MA, Kappetein AP, Morice MC, Colombo A, Dawkins K, et al. The SYNTAX score: an angiographic tool grading the complexity of coronary artery disease. *EuroIntervention.* 2005;1(2):219–227.
14. Ashfaq F, Goel PK, Siddiqi S, Nazir A, Yaqub M. Lipoprotein(a) and SYNTAX score association with severity of coronary artery atherosclerosis. *J Saudi Heart Assoc.* 2012;24(4):257-264.

Figures

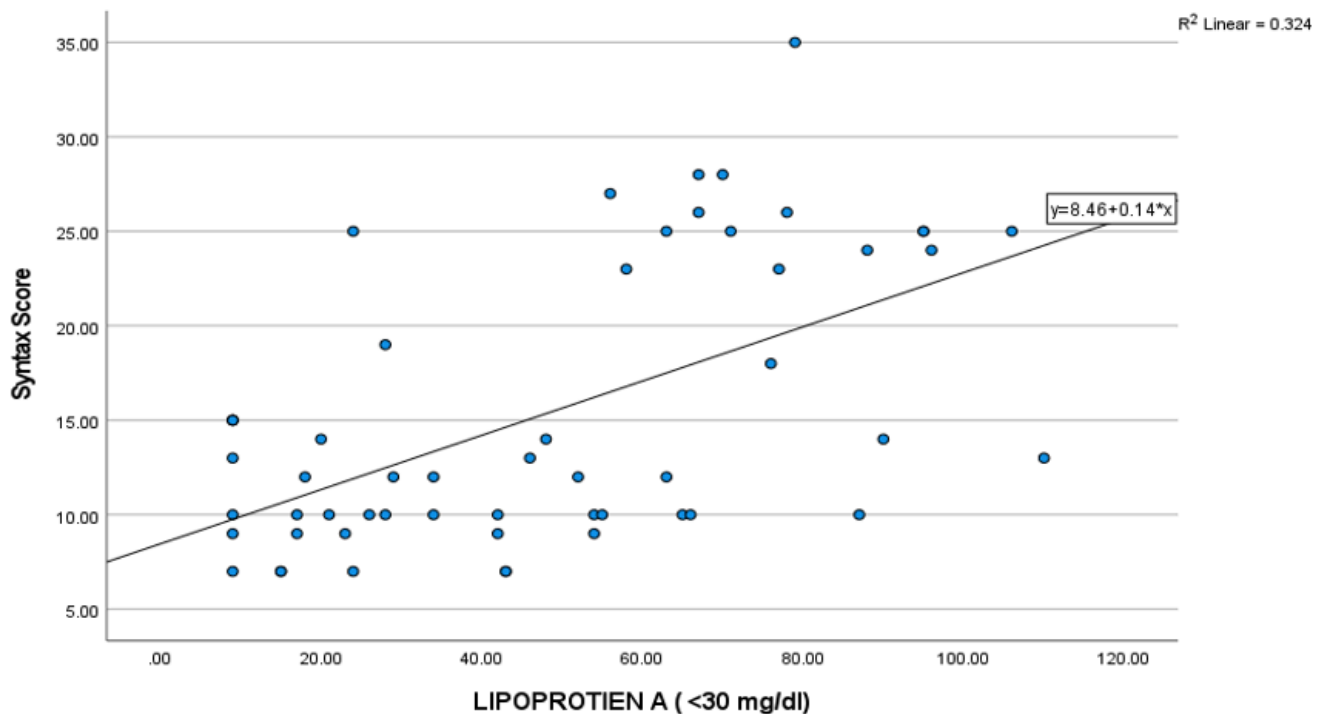


Figure 1

Scatter plot demonstrating a moderate positive correlation between Lipoprotein(a) concentration and SYNTAX score (Spearman's correlation coefficient $r = 0.540$, $p < 0.001$). Higher Lipoprotein(a) levels were associated with increased angiographic complexity and severity of coronary artery disease

Figure 1. Correlation between serum Lipoprotein(a) levels and SYNTAX score in young adults presenting with acute coronary syndrome. Scatter plot demonstrating a moderate positive correlation between Lipoprotein(a) levels and SYNTAX score (Spearman's correlation coefficient $r = 0.540$, $p < 0.001$), indicating an association between elevated Lipoprotein(a) concentrations and increased angiographic severity of coronary artery disease.