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Lipoprotein(a) and Coronary Severity: A Cross-Sectional Analysis in Cameroon

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Abstract

Background: Lipoprotein(a) [Lp(a)] is an independent cardiovascular risk factor involved in the occurrence of acute coronary syndromes. However, its relationship with the anatomical severity of coronary lesions remains poorly documented in sub-Saharan Africa. **Objective:** To assess the association between elevated lipoprotein(a) levels and the severity of coronary lesions in patients admitted for acute coronary syndrome in Yaoundé. **Methods:** We carried out an observational cross-sectional analytical study using data from the Yaoundé interventional cardiology registry (DéRICA) over a three-year period (November 2022-November 2025). Patients with acute coronary syndrome, significant coronary lesions (≥50%), and elevated lipoprotein(a) levels (≥50 mg/dL) were included. Coronary lesion severity was assessed using the ACC/AHA classification and grouped as non-severe (A, B1) or severe (B2, C). Statistical analyses were performed using logistic regression, with $p < 0.05$ considered statistically significant. **Results:** Among patients with elevated lipoprotein(a), a high proportion presented with severe coronary lesions (B2/C). Severe lesions were significantly associated with triple-vessel disease, Heart Team-based therapeutic decision-making, and complex treatment strategies. A near-significant association was observed with left and balanced coronary dominance. Traditional cardiovascular risk factors were not significantly associated with lesion severity. **Conclusion:** In patients with elevated lipoprotein(a), severe coronary lesions are frequent and reflect a complex anatomical pattern of coronary artery disease. Lipoprotein(a) may serve as a relevant biological marker of coronary lesion severity. Larger multicenter studies are warranted to confirm these findings.

Keywords

Lipoprotein(a), Acute Coronary Syndrome, Coronary Lesion Severity, Coronary Angiography, Sub-Saharan Africa

1. Introduction

Cardiovascular diseases represent the leading cause of mortality worldwide, resulting in nearly 19.8 million annual deaths, accounting for approximately one-third of all global deaths [1]. Among these deaths, the majority are attributable to ischemic events, particularly acute coronary syndromes.

Lipoprotein(a) [Lp(a)] is a lipoprotein particle closely related to low-density lipoproteins, characterized by the covalent bonding of an apolipoprotein(a) to an apo B-100 molecule; this structure confers atherogenic, pro-inflammatory, and prothrombotic properties [2] [3]. Plasma concentrations of Lp(a) are predominantly genetically determined and remain relatively stable throughout the lifespan [4].

Epidemiological, genetic, and association studies have demonstrated that elevated Lp(a) concentrations constitute an independent and causal risk factor for atherosclerotic cardiovascular disease, including coronary artery disease, stroke, and aortic valve stenosis [5] [6]. Furthermore, Mendelian randomization analyses reinforce the relationship between high Lp(a) levels and an increased risk of cardiovascular events, independent of LDL-C cholesterol levels [5].

Several clinical guidelines recognize the clinical utility of a one-time measurement of Lp(a) concentrations in adults to improve cardiovascular risk stratification and consider levels ≥50 mg/dL to be associated with an increased risk of cardiovascular events [7]. Despite these advancements, data regarding the relationship between elevated Lp(a) levels and the anatomical severity of coronary lesions on angiography remain limited, particularly in resource-limited settings.

In this context, the objective of this study was to evaluate the association between elevated Lp(a) levels and the severity of coronary lesions, as defined by the ACC/AHA angiographic classification, in patients managed for coronary artery disease at a reference center in Yaoundé.

2. Methods

Study design and setting

We conducted an analytical cross-sectional observational study using data from the Yaoundé interventional cardiology registry, known as DéRICA (yaounDé Registry of Interventional Cardiology Achievements). This registry prospectively includes all patients admitted to the cardiac catheterization laboratory of the Cardiovascular Explorations Department at the Yaoundé General Hospital. This facility houses Cameroon's first and only cardiac catheterization laboratory, which has

been operational since November 2022 and is operated by an interventional cardiologist.

The study covered a three-year period, from November 8, 2022, to November 8, 2025.

Study population

All patients were included consecutively and exhaustively based on the following criteria:

* Admitted for acute coronary syndrome;
* Underwent diagnostic coronary angiography;
* Presented with significant coronary lesions, defined as stenosis ≥ 50% by quantitative coronary analysis (QCA);
* Had available lipoprotein(a) [Lp(a)] measurements.

For the present analysis, only patients with elevated lipoprotein(a), defined by a threshold ≥50 mg/dL, were included.

Diagnostic criteria for acute coronary syndrome

Acute coronary syndrome (ACS) was defined according to the criteria of the Fourth Universal Definition of Myocardial Infarction and current international guidelines. STEMI was diagnosed in the presence of persistent ST-segment elevation in ≥2 contiguous leads associated with elevated cardiac troponin levels. NSTEMI was defined by elevated cardiac troponin levels without persistent ST-segment elevation but with ischemic symptoms and/or dynamic ECG changes. Unstable angina was defined as ischemic symptoms suggestive of myocardial ischemia without elevation of cardiac biomarkers.

Patients without Lp(a) measurements, those with incomplete angiographic data, and cases classified as non-interpretable (NI) according to the angiographic classification were excluded from the lesion severity analysis.

The sampling method was non-probabilistic, consecutive, and exhaustive.

Coronary angiography procedure

Examinations were performed in the cardiac catheterization laboratory equipped with a SIEMENS Artis One angiograph. Coronary angiography was conducted according to standard practices, via radial or femoral access, with visual analysis supplemented by quantitative coronary analysis (QCA).

Assessment of coronary lesion severity

The anatomical severity of coronary lesions was assessed using the American College of Cardiology/American Heart Association (ACC/AHA) classification, distinguishing between types A, B1, B2, and C.

For analytical purposes, lesions were grouped as follows:

- Non-severe lesions: types A and B1;
- Severe lesions: types B2 and C.

When multiple coronary lesions were present in the same patient, classification at the patient level was based on the lesion with the highest anatomical complexity (worst-lesion approach).

Angiographic grading according to the ACC/AHA classification was per-

formed by the interventional cardiologist who conducted the procedure. In cases of uncertainty, angiographic images were reviewed to ensure classification consistency.

All procedures and angiographic assessments were performed by a single experienced interventional cardiologist.

Study variables

The collected variables included:

- Sociodemographic data;
- Cardiovascular risk factors;
- Clinical characteristics of the acute coronary syndrome;
- Biological data, including lipoprotein(a) levels;
- Angiographic characteristics (number of vessels involved, coronary dominance, lesion severity);
- Therapeutic strategies (Heart Team discussion, percutaneous coronary intervention).

The primary endpoint was the presence of severe coronary lesions (B2/C).

Statistical analysis

Data were analyzed using SPSS software, version 26.0. Qualitative variables were described using frequencies and percentages, while quantitative variables were presented as means ± standard deviation or medians, depending on their distribution.

Given the small sample size, multivariable logistic regression was not performed to avoid model overfitting. Associations between variables and lesion severity were therefore explored using univariate analyses.

Results were expressed as odds ratios (OR) with their 95% confidence intervals. A p-value < 0.05 was considered statistically significant.

Ethical considerations

All patients provided written informed consent prior to their inclusion in the registry. The study received approval from the Ethics and Pharmacovigilance Committee of the Yaoundé General Hospital, in accordance with the ethical principles of the Declaration of Helsinki.

3. Results

During the study period, 115 patients were managed at the center. Among them, 38 patients presented with elevated lipoprotein(a) and constituted the target population. Twenty-three patients had interpretable angiographic data classifiable according to the ACC/AHA system (A, B1, B2, and C) and were included in the analysis, while the 14 remaining patients were not included due to non-interpretable angiographic findings.

Fourteen patients were excluded because their coronary angiography was considered non-interpretable for lesion classification. In several cases, the angiographic findings showed mixed or diffuse coronary lesions, with varying degrees of stenosis around the 50% threshold, which made it difficult to clearly identify or

classify a target lesion according to the ACC/AHA classification.

A comparison of baseline characteristics between included and excluded patients showed no significant differences in age, cardiovascular risk factors, or Lp(a) levels (Appendix Table A1).

Among the 23 patients included in the final analysis, 16 (69.6%) presented with ST-segment elevation myocardial infarction (STEMI), 5 (21.7%) with non-ST-segment elevation myocardial infarction (NSTEMI), and 2 (8.7%) with unstable angina.

General Characteristics

The mean age of the included patients was 63.42 ± 11.46 years, with a range of 27 to 82 years. A male predominance was observed, representing 87% of the population. The distribution by age group and sex did not differ significantly between patients with severe lesions (B2/C) and those with non-severe lesions (A/B1). The distribution of ACS subtypes did not differ significantly between patients with severe and non-severe coronary lesions (Fischer exact test) (Table 1).

Table 1. Sociodemographic characteristics of patients with ACS: comparison between angiographic severe and non-severe lesions.

Variables	Total Cohort (N= 23)	Severe Lesions (B2.C) n = 12	Non Severe Lesions (A. B1) n = 11	p-Value	OR (95% CI)
Mean age ± SD	63.42 ± 11.46	62.17 ± 7.83	63.27 ± 8.78	0.77	Mean diff: 1.25 (−7.58 - 10.08)
Age groups (years)					
<40	0	0	0		
[41 - 50]	1 (4.3)	0	1 (9.1)	0.478	
[51 - 60]	9 (39.1)	6 (50)	3 (27.3)	0.400	2.67 (0.47 - 15.25)
[61 - 70]	10 (43.5)	5 (41.7)	5 (45.5)	1.00	0.86 (0.16 - 4.47)
>70	3 (13)	1 (8.3)	2 (18.2)	0.590	0.41 (0.03 - 5.28)
Sex					
Male	20 (87)	10 (83.3)	10 (90.9)	1.00	0.50 (0.04 - 6.44)
Female	3 (13)	2 (16.7)	1 (9.1)		2.00 (0.16 - 25.76)
ACS subtype					
STEMI	16 (69.6)	8 (66.7)	8 (72.7)	1.00	0.75 (0.13 - 4.49)
NSTEMI	5 (21.7)	2 (16.7)	3 (27.3)	0.640	0.53 (0.07 - 4.01)
Unstable angina	2 (8.7)	2 (16.7)	0	0.478	

SD: Standard Deviation; **ACS**: Acute coronary Syndrom; NSTEMI: Non ST Elevation Myocardial Infarction; STEMI: ST Elevation Myocardial Infarction.

Cardiovascular risk factors were highly prevalent in both groups. Hypertension was present in 82.6% of patients, type 2 diabetes in 34.8%, dyslipidemia in 73.9%, and tobacco use in 26.1%. No statistically significant differences were observed between the two groups regarding medical history, associated heart diseases,

comorbidities, impaired left ventricular ejection fraction, or medical treatments prior to admission (Table 2).

Table 2. Cardiovascular risk factors, medical history, and comorbidities in coronary patients according to lipid status.

Variables	Total cohort (N = 23)	Severe lesions (B2, C) (N = 12)	Non-severe lesions (A, B1) N = 11	p value	OR (95% CI)
Medical History					
History of CAD	1 (4.3)	1 (8.3)	0	1.00	
Hypertension	19 (82.6)	10 (83.3)	9 (81.8)	1.00	1.11 (0.13 - 9.61)
Type 2 Diabetes	8 (34.8)	5 (41.7)	3 (27.3)	0.667	1.91 (0.33 - 11.01)
Dyslipidemia	17 (73.9)	10 (83.3)	7 (63.6)	0.371	2.86 (0.41 - 20.14)
Tobacco use	6 (26.1)	2 (16.7)	4 (36.4)	0.371	0.35 (0.05 - 2.47)
Heart failure	4 (17.4)	2 (16.7)	2 (18.2)	1.00	0.90 (0.11 - 7.78)
Heart Disease					
Hypertensive heart disease	12 (52.2)	8 (66.7)	4 (36.4)	0.146	3.50 (0.63 - 19.50)
Ischemic heart disease	14 (60.9)	6 (50)	8 (72.7)	0.400	0.37 (0.07 - 2.15)
Mixed heart disease	5 (21.7)	3 (25)	2 (18.2)	1.00	1.5 (0.20 - 11.23)
Comorbidities					
HIV Infection	3 (13)	1 (8.3)	2 (18.2)	0.590	0.41 (0.03 - 5.28)
Impaired LVEF (< 50%)	11 (47.8)	6 (50.0)	5 (45.5)	1.00	1.2 (0.23 - 6.19)
Medical therapy prior to admission					
DAPT	21 (91.3)	12 (100)	9 (81.8)	0.217	
OMT	15 (65.2)	7 (58.3)	8 (72.7)	0.667	0.53 (0.09 - 3.03)

CAD: Coronary Artery Disease (Antécédents coronaires), **LVEF:** Left Ventricular Ejection Fraction (FEVG), **DAPT:** Dual Antiplatelet Therapy (Double anti-agrégation plaquettaire), **OMT:** Optimal Medical Therapy, **OR (95% CI):** Odds Ratio (95% Confidence Interval).

From an angiographic perspective, the severity of coronary lesions was defined by the presence of type B2 or C lesions. Twelve patients (52.2%) presented with severe lesions, compared to eleven patients (47.8%) with non-severe lesions. Three-vessel disease was significantly more frequent in patients with severe lesions (66.7% vs. 18.2%; p = 0.036), with an odds ratio of 9.00 (95% CI: 1.28 - 63.03).

Regarding coronary dominance, a difference approaching the threshold of statistical significance was observed for left and balanced dominance. Left dominance was found in 8.3% of patients with severe lesions compared to 45.5% in those with non-severe lesions (p = 0.069), while balanced dominance was observed in 50.0% of patients with severe lesions compared to 9.1% in those with non-severe lesions (p = 0.069) (Table 3).

Table 3. Time to presentation and angiographic characteristics according to coronary lesion severity.

Variables	Total cohort (N = 23)	Severe lesions (B2, C) (N = 12)	Non-severe lesions (A, B1) (N= 11)	p-value	OR (95% CI)
Time to presentation					
<24 hrs	2 (8.7)	1 (8.3)	1 (9.1)	1.00	0.9 (0.05 - 16.54)
[1 - 30] days	14 (60.9)	6 (50)	8 (72.7)	0.400	0.38 (0.07 - 2.14)
[1 - 3] months	2 (8.7)	1 (8.3)	1 (9.1)	1.00	0.9 (0.05 - 16.54)
>3 months	5 (21.7)	4 (33.3)	1 (9.1)	0.317	5.00 (0.46 - 54.0)
Coronary Dominance					
Right	10 (43.5)	5 (41.7)	5 (45.5)	1.00	0.86 (0.16 - 4.47)
Left	6 (26.1)	1 (8.3)	5 (45.5)	0.069	0.11 (0.01 - 1.16)
Balanced	7 (30.4)	6 (50)	1 (9.1)	0.069	10.0 (0.96 - 104.5)
Vessel involvement					
Single-vessel disease	11 (47.8)	4 (33.3)	7 (63.6)	0.146	0.28 (0.05 - 1.59)
Two-vessel disease	2 (8.7)	0	2 (8.7)	0.217	
Three-vessel disease	10 (43.5)	8 (66.7)	2 (18.2)	**0.036**	9.00 (1.28 - 63.03)

Regarding therapeutic strategies, percutaneous coronary intervention (PCI) was performed in 52.2% of patients. It was significantly more frequent in patients with non-severe lesions compared to those with severe lesions (90.9% vs. 16.7%; $p < 0.001$), with an odds ratio of 0.02 (95% CI: 0.002 - 0.26). Conversely, Heart Team discussions involved exclusively patients with severe lesions (58.3% vs. 0%; $p = 0.005$). These therapeutic decisions reflect the anatomical complexity rather than being determinants of lesion severity.

Coronary artery bypass graft (CABG) surgery was performed in three patients, all were in the severe lesions sub group, though this did not reach statistical significance (Table 4).

Table 4. Therapeutic strategies and revascularization modalities according to coronary lesion severity.

Variables	Total cohort (N = 23)	Severe lesions (B2, C) (N = 12)	Non-severe lesions (A, B1) (N = 11)	p-value	OR (95% CI)
Percutaneous Coronary Intervention (PCI)	12 (52.2)	2 (16.7)	10 (90.9)	**<0.001**	0.02 (0.002 - 0.26)
Optimal Medical Therapy (OMT)	0	0	0		
Coronary Artery Bypass Graft (CABG)	3 (13)	3 (25.0)	0	0.217	
Heart Team Discussion	7 (30.4)	7 (58.3)	0	**0.005**	
Other management strategies	1 (4.3)	0	1 (9.1)	0.478	

4. Discussion

In this study, we observed that among patients with elevated lipoprotein(a), a significant proportion presented with severe coronary lesions (B2/C) on angiography.

This observation suggests a relationship between elevated Lp(a) levels and a more complex anatomical phenotype of coronary artery disease.

Importantly, the distribution of ACS subtypes did not differ significantly between patients with severe and non-severe lesions, suggesting that the observed anatomical complexity was unlikely to be solely driven by the type of clinical presentation.

Several published data confirm an association between elevated Lp(a) levels and the severity or complexity of coronary lesions. For instance, a study conducted in a large cohort of patients with acute coronary syndrome showed that Lp(a) levels > 50 mg/dL were significantly associated with a high SYNTAX score, used as a measure of lesion complexity, independent of traditional cardiovascular risk factors (OR = 1.54; p = 0.02) [8]. Similarly, another study in patients with stable coronary artery disease found a positive correlation between Lp(a) levels and the SYNTAX score, indicating that higher Lp(a) levels were associated with more severe coronary lesions ($p < 0.001$) [9]. A cross-sectional study also reported a significant positive correlation between Lp(a) and lesion complexity (SYNTAX score) ($r = 0.33$; $p = 0.004$) [10].

The results observed in the literature are consistent with the pathophysiological hypothesis that elevated lipoprotein(a) is associated with a more aggressive progression of atherosclerosis, reflected by a greater plaque burden and more complex coronary lesions. Indeed, elevated Lp(a) levels have been associated with significant progression of coronary artery disease measured by quantitative angiographic scores such as the Gensini score ($p < 0.001$), highlighting a link between Lp(a) and the advancement of coronary atheroma [11].

The absence of a significant association between classic cardiovascular risk factors and lesion severity in our cohort may reflect the inherently genetic nature of Lp(a), which confers risk independent of traditional clinical factors [12]. Furthermore, research in patients with familial hypercholesterolemia has shown that Lp(a) levels were independently associated with the severity of coronary atherosclerosis measured by the Gensini score ($p = 0.006$) [13].

The observation of an association between more complex therapeutic strategies (Heart Team and PCI) and lesion severity reflects the immediate clinical implications of anatomical complexity. The study distinguished anatomical severity from subsequent therapeutic decisions. Heart Team consultation and PCI implementation were considered consequences of lesion complexity and were not included as explanatory variables in the regression analysis.

Study Limitations

Our study has several important limitations. The small sample size limits statistical power, and the single-center design restricts the generalizability of the results. Furthermore, the absence of a control group with normal Lp(a) levels prevents a direct comparison between patients with and without elevated Lp(a). Also, the use of the ACC/AHA classification, without the application of quantitative angiographic scores such as SYNTAX or Gensini, may limit the granularity of the assess-

ment of lesion severity, although these scores were used in the cited studies to demonstrate similar results [8]. The ACC/AHA classification, while clinically practical, may be less suited for patients presenting with diffuse or heterogeneous coronary patterns, which contributed to the proportion of non-interpretable cases in our cohort. Finally, With only 23 patients included, the statistical power to detect significant associations remains limited. In addition, multivariable regression analysis could not be reliably performed due to the low number of events, which increases the risk of model overfitting. Therefore, the findings should be interpreted cautiously and confirmed in larger studies.

In conclusion, our results, in agreement with existing literature, suggest that elevated lipoprotein(a) levels are associated with a more severe anatomical burden and complex coronary lesions. This highlights the importance of careful angiographic evaluation in patients with high biological risk, as well as the need for larger studies to better define the impact of Lp(a) on the severity of coronary artery disease.

5. Conclusions

This study, derived from the Yaoundé interventional cardiology registry, demonstrates that within a population with high lipoprotein(a) concentrations, severe coronary lesions (defined by ACC/AHA types B2 and C) are frequent and are associated with angiographic and therapeutic profiles reflecting more complex coronary artery disease. Three-vessel disease, Heart Team discussion, and the observed revascularization strategies reflect this anatomical complexity.

Although limited by a small sample size and the absence of quantitative angiographic scores, this study suggests that lipoprotein(a) may constitute a relevant biological marker of anatomical severity in coronary artery disease, independent of traditional cardiovascular risk factors. These results reinforce the clinical utility of integrating lipoprotein(a) assessment in risk stratification of coronary patients, particularly in resource-limited settings.

Multicenter studies, including larger cohorts and utilizing validated angiographic scores, will be necessary to confirm these observations and clarify the role of lipoprotein(a) in risk stratification and therapeutic guidance for patients with coronary artery disease.

Authors' Contributions

All authors contributed to the conception, analysis, interpretation, and writing of this work. Therefore, all authors assume responsibility for all aspects of the work.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Appendix

Table A1. Comparison of baseline characteristics between included and excluded patients.

Variable	Included (n = 23)	Excluded (n = 14)	p-value	Distribution (Comparison test)
Age (mean ± SD)	62.70 ± 8.12	63.43 ± 15.49	0.872	Normal distribution (Student's t-test)
Male sex n (%)	20 (87.0)	10 (71.4)	0.390	Fisher's exact test
Hypertension n (%)	19 (82.6)	11 (78.6)	1.00	Fisher's exact test
Diabetes n (%)	8 (34.8)	4 (28.6)	1.00	Fisher's exact test
STEMI n (%)	16 (69.6)	5(35.7)	0.044	Chi-square test
LP(a) level (median (IQR))	76.8 (59.7 - 82.5)	57 (51.1 - 80.47)	0.062	Non-normal (Mann-Whitney test)