Research Square

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# Lipoprotein-associated phospholipase A2 and Its Association with Acute Coronary Syndrome: A Comprehensive Analysis of the Correlation Between Coronary Artery Disease Severity and Post-percutaneous coronary intervention Cardiovascular Endpoint Events

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# Abstract

Background: As an emerging inflammatory marker involved in atherosclerosis, Lp-PLA2 plays an important role in the occurrence and development of ACS.

Objective: This study aims to investigate the correlation between Lp-PLA2 levels and both the severity of coronary artery lesions in ACS patients and the incidence of cardiovascular endpoint events following PCI.

Patients and methods:Patients initially diagnosed with ACS who underwent CAG and PCI at the First People's Hospital of Chenzhou City between January 2019 and July 2022 were enrolled in the study and subsequently followed up for a 36-month period.

Result : 1.In the classification of coronary artery lesion severity based on the Gensini score, after adjusting for confounding factors through binary logistic regression analysis, it was demonstrated that elevated serum Lp-PLA2 concentration constitutes an independent risk factor for the severity of coronary artery lesions in patients with ACS,(OR=2.114).2.In the stratification analysis based on cardiovascular endpoint events, after adjusting for confounding factors through Cox proportional hazards regression analysis, elevated Lp-PLA2 levels were identified as an independent risk factor for cardiovascular endpoint events in ACS patients following PCI (RR=2.08).3.In the stratification based on Lp-PLA2 concentration levels, subsequent to multivariate adjustment through binary logistic regression analysis, it was demonstrated that both smoking status and LDL-C levels exhibited significant positive associations with Lp-PLA2 concentration, respectively.

**Conclusion:** Lp-PLA2 concentration serves as an independent prognostic indicator for both the severity of coronary artery lesions in ACS patients and the incidence of cardiovascular endpoint events following PCI.


# Introduction

ACS represents a spectrum of clinical manifestations resulting from acute myocardial ischemia. Atherosclerosis (AS) constitutes the primary pathological foundation of ACS. Recent scientific advancements have redefined atherosclerosis as a lipid-driven inflammatory disorder, wherein pro-inflammatory mediators play a pivotal role in determining the pathogenesis, disease progression, and ultimate clinical outcomes of atherosclerosis[1-2].Inflammation plays a pivotal role throughout the entire process of atherosclerotic plaque development, encompassing initial plaque formation, plaque destabilization, and eventual plaque rupture[3].This phenomenon results in a substantial elevation in the risk of adverse cardiovascular events. Lp-PLA2 has progressively garnered significant attention as a novel inflammatory biomarker implicated in atherosclerosis.This enzyme is capable of catalyzing the hydrolysis of oxidized low-density lipoprotein (ox-LDL), yielding two biologically active metabolites: lysophosphatidylcholine (Lyso-PC) and oxidized free fatty acids (oxFFA). These metabolites play a pivotal role in triggering an inflammatory cascade during the pathogenesis of atherosclerosis[4-

[5].However, the activated inflammatory cells can enhance the production of Lp-PLA2, leading to increased deposition of Lyso-PC and oxFFA beneath the arterial intima, thereby promoting the formation and progression of plaque lipid cores[6-7].Isabel et al. demonstrated that the concentration of Lp-PLA2 within atherosclerotic plaques exhibits a significant correlation with plaque stability[8].The elevation in Lp-PLA2 concentration induces the expansion of its lipid pool, resulting in the attenuation of the fibrous membrane, thereby exacerbating plaque instability and elevating the risk of cardiovascular events.Empirical studies have demonstrated a positive correlation between elevated plasma Lp-PLA2 concentrations and the severity of coronary atherosclerosis[9].The genetic polymorphism of Lp-PLA2 has been demonstrated to be significantly correlated with coronary heart disease (CHD). Specifically, this polymorphism influences the expression levels of Lp-PLA2, consequently contributing to the pathogenesis and progression of CHD. Furthermore, Lp-PLA2 serves as a valuable biomarker for cardiovascular risk assessment and potentially represents a novel therapeutic target for cardiovascular diseases[10].In preclinical animal studies, the inhibition of Lp-PLA2 concentration demonstrates therapeutic potential in alleviating atherosclerosis[11].Nevertheless, empirical findings substantiate that Lp-PLA2 serves as a biomarker for oxidative stress and inflammation, rather than functioning as an independent risk factor for cardiovascular diseases[12].A comprehensive randomized clinical trial demonstrated that the therapeutic efficacy of darapladib in inhibiting Lp-PLA2 concentration among patients with ACS remains inconclusive regarding mid-term prognostic outcomes and the attenuation of vulnerable plaque progression[13].In light of these contentious findings, we undertook a comprehensive retrospective clinical investigation to elucidate the prognostic significance of Lp-PLA2 levels in assessing the severity of coronary artery lesions in ACS patients, as well as its predictive value for cardiovascular endpoint events following PCI. This study provides substantial evidence to inform clinical decision-making and therapeutic strategies for ACS management.

# 1 Materials and Methods

1.1 **Object of study** From January 2019 to July 2022, a cohort of 174 patients with initial diagnosis of ACS who underwent coronary angiography and PCI was identified at the First People's Hospital of Chenzhou City.The diagnosis of ACS is established in accordance with the "Emergency Rapid Diagnosis and Treatment Guidelines for Acute Coronary Syndrome (2019)".[14]The follow-up duration spanned 36 months.Exclusion criteria: 1. Coexisting cardiovascular diseases (excluding complications of ACS). 2. Severe hepatic or renal dysfunction or failure of vital organs; 3. Concurrent infectious or autoimmune diseases; 4. Psychiatric disorders, malignant neoplasms, hematological disorders, or related conditions; 5. Patients with incomplete clinical data or inability to complete follow-up.

1.2 **Grouping** Initially, the study participants were stratified into two distinct cohorts according to the median Gensini score: the mild lesion group comprising 87 cases (12-86 points) and the severe lesion group consisting of 87 cases (88-212 points). Subsequently, based on the occurrence of cardiovascular endpoint events, the participants were categorized into the non-event group (n=131) and the event group (n=43). Ultimately, utilizing the plasma concentration of Lp-PLA2 as a criterion, the cohort was further

divided into the low Lp-PLA2 concentration group (n=87) and the high Lp-PLA2 concentration group.The correlation between Lp-PLA2 and established risk factors for coronary heart disease was analyzed. For comprehensive details, please refer to the research flowchart presented in Table 1.

1.3 **Method** Comprehensive demographic and clinical baseline data were meticulously documented for the selected ACS patients, encompassing age, gender, and pertinent medical history, including body mass index (BMI), hypertension, type 2 diabetes mellitus, and smoking status. Venous blood samples were collected from all ACS patients immediately upon hospital admission for comprehensive laboratory analyses, including cardiac troponin I (cTNI), complete blood count, hepatic function panel, renal function panel, lipid profile, and Lp-PLA2.The plasma Lp-PLA2 concentration was quantified using enzyme-linked immunosorbent assay (ELISA). The ELISA kit was procured from Kangerke Biotechnology Co.Ltd. (Tianjin, China). The experimental procedures were meticulously executed in strict adherence to the manufacturer's protocol to ensure quality control.Coronary angiography was conducted through the radial artery approach, utilizing standard multi-projection radiographic imaging. The coronary angiography findings were analyzed and the Gensini score was calculated by two board-certified interventional cardiologists. Following the angiographic assessment, the lesion was precisely identified, and PCI was subsequently performed. Concurrently, all pertinent procedural details of the PCI were meticulously documented.

1.4 **Follow-up method** Telephone follow-up, outpatient follow-up, and inpatient medical record retrieval were implemented. Throughout the follow-up period, comprehensive clinical data were collected, including patients' general clinical status, post-discharge medication regimens, as well as the type and time of cardiovascular endpoint events.The definition of cardiovascular endpoint events is consistent with the "2014ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery"[15]：1. Cardiovascular mortality.2. Non-cardiovascular mortality.3. Recurrent myocardial infarction.4. Hospitalization due to unstable angina pectoris.5. Repeat PCI.6. Peripheral vascular intervention.7. Heart failure events.8. Stent thrombosis.9. Transient ischemic attack (TIA) and stroke.Definition of the non-event group: No cardiovascular endpoint events were observed during the follow-up period of this study.Definition of the event group: At least one cardiovascular endpoint event occurred.

1.5 **Statistical treatment** All data were analyzed utilizing SPSS 27.0 software. The measurement data were presented as mean values±standard deviation. The ROC curve analysis was employed to evaluate the predictive value of Lp-PLA regarding the severity of coronary artery lesions and post-PCI cardiovascular endpoint events.The homogeneity of variance was assessed through the Levene's F test. an independent samples t-test was employed to compare the two groups. Categorical data were presented as frequencies (percentages). Intergroup comparisons were performed using the chi-square test, while multivariate analyses were conducted through binary logistic regression or Cox proportional hazards regression. A P-value of less than 0.05 was deemed statistically significant.

# 2 Results

2.1 Among patients with ACS, the ROC curve analysis demonstrated a significant association between Lp-PLA2 levels and the severity of coronary artery lesions (Figure 1). The AUC was 0.625 (95%CI: 0.542-0.709), with an optimal cut-off value of 162.75, yielding a sensitivity of 57.5% and a specificity of 66.7%. Based on the ROC curve analysis, patients were stratified into low-concentration and high-concentration groups according to their Lp-PLA2 levels.The comparative analysis of clinical baseline characteristics across the quantile groups based on Gensini scores (Table 2) demonstrated statistically significant differences in patients with elevated Lp-PLA2 concentrations, smoke, type 2 diabetes mellitus, and albumin levels (P<0.05).Following adjustment for smoke, type 2 diabetes, and albumin levels via binary logistic regression analysis (Table 3), elevated Lp-PLA2 concentrations demonstrated a statistically significant positive association with the severity of coronary artery disease (P=0.024), yielding an odds ratio (OR) of 2.114 (95%CI: 1.103-4.048).

2.2 A cohort of 174 ACS patients with complete datasets was prospectively followed for 36 months, demonstrating a mean follow-up duration of 30.59±10.73 months. During the observation period, 43 cardiovascular endpoint events were documented (Table 4), including: 1 case of cardiovascular mortality; 0 cases of non-cardiovascular mortality; 5 cases of recurrent myocardial infarction; 16 cases of hospitalizations due to unstable angina pectoris; 12cases of requiring repeat PCI procedures; 0 cases of peripheral vascular intervention; 9 cases of heart failure; and 0 cases of cerebrovascular accident.Throughout the follow-up period, all patients with ACS adhered to the "Emergency Rapid Diagnosis and Treatment Guidelines for Acute Coronary Syndrome (2019)" for antiplatelet therapy, complied with the "Expert Consensus on Clinical Pathways for Lipid Management in Patients with Acute Coronary Syndrome (2020)" [16]for lipid-lowering treatment, and all smoking patients strictly abstained from tobacco use.The ROC curve analysis of Lp-PLA2 in relation to cardiovascular endpoint events post-PCI among ACS patients (Figure 2) demonstrated an AUC of 0.622, with an optimal cut-off value of 170.67, yielding a sensitivity of 55.8% and a specificity of 69.5%. Based on these ROC curve findings, patients were stratified into low-concentration and high-concentration groups according to their Lp-PLA2 levels.The comparative analysis of baseline clinical characteristics between the non-event and event groups (Table 5) demonstrated a significantly higher prevalence of patients with elevated levels of smoke, TC, lLDL-C, and Lp-PLA2 in the event group compared to the non-event group (P<0.05). Cox proportional hazards regression analysis (Table 6, Figure 3) revealed that elevated Lp-PLA2 levels were significantly associated with an increased risk of cardiovascular endpoint events following PCI in patients with ACS (P=0.023), with a RR of 2.08 (95%CI:1.106-3.922).

2.3 Among patients with ACS, the comparative analysis of baseline clinical characteristics between the low-concentration Lp-PLA2 group and the high-concentration Lp-PLA2 group (Table 7) demonstrated that smoke, TC, and LDL-C levels were significantly elevated in the high-concentration group compared to the low-concentration group (P< 0.05).Following adjustment for the effects of smoke, TC, LDL-C through binary logistic regression analysis (Table 8), smoke, LDL-C, and elevated Lp-PLA2 concentration demonstrated significant positive correlations, with corresponding odds ratios (OR) of 1.909 (95% CI: 1.008-3.619, P=0.047) and 2.439 (95% CI: 1.033-5.780, P=0.042), respectively.

# 3 Discussion

With the accelerated development of China's economy and society, the proportion of the aging population has been progressively increasing, accompanied by continuous enhancements in living standards and significant transformations in lifestyle patterns. Despite the ongoing advancements in medical technology, the incidence and mortality rates of CHD within the Chinese population continue to exhibit a gradual upward trend, remaining inadequately controlled. The pathogenesis and progression of coronary heart disease are associated with multiple factors.Lp-PLA2, a platelet-activating factor acetylhydrolase initially identified in 1980, has emerged as a significant inflammatory biomarker in contemporary medical research. This enzyme is recognized as one of the pivotal cardiovascular biomarkers that demonstrate a strong correlation with elevated risks of CHD.In atherosclerotic lesions, Lp-PLA2 is predominantly synthesized and expressed by macrophages, lymphocytes, monocytes, and mast cells. The pro-atherogenic effect of Lp-PLA2 primarily manifests through the hydrolysis of ox-LDL into two bioactive metabolites: Lyso-PC and oxFFA. These metabolites play crucial roles in the pathogenesis and progression of atherosclerosis, initiating an inflammatory cascade reaction during the atherosclerotic process[4-5].However, the activation of inflammatory cells can induce increased production of Lp-PLA2, thereby establishing a self-perpetuating cycle. This pathological process ultimately results in enhanced deposition of Lyso-PC and oxFFA beneath the arterial intima, consequently accelerating the progression of the atherosclerotic plaque lipid core[6-7].Consequently, Lp-PLA2 is situated at the nexus of lipid metabolism and inflammatory pathways. Through its involvement in dyslipidemia regulation and the facilitation of vascular inflammatory processes, it contributes to the pathogenesis and progression of atherosclerosis, ultimately culminating in the onset of ACS[17-18].

This study demonstrated that in patients with ACS, the utilization of the Gensini score for assessing the severity of coronary artery lesions revealed that Lp-PLA2 possesses significant predictive value for coronary artery lesion severity. The ROC curve analysis yielded an area under the curve (AUC) of 0.625, with an optimal cut-off value of 162.75, demonstrating a sensitivity of 57.5% and a specificity of 66.7%.Patients exhibiting elevated concentrations of Lp-PLA2 in the severe coronary artery lesion group demonstrated significantly higher levels compared to those in the mild lesion group. Following further adjustment for confounding factors, including smoke, type 2 diabetes mellitus, and albumin levels through binary logistic regression analysis, elevated Lp-PLA2 concentrations remained positively associated with the severity of coronary artery lesions in ACS, as quantified by the Gensini score (OR: 2.114).The Gensini score represents an early-established scoring system for coronary artery lesions. This system comprehensively evaluates the severity of coronary artery lesions by incorporating three key parameters: the number of lesions, their anatomical locations, and the degree of stenosis, thereby providing a quantitative assessment of coronary artery disease severity.Through postmortem analysis, Kolodgie et al. demonstrated that in individuals who succumbed to coronary events, the concentration of Lp-PLA2 in plaque tissue exhibited a positive correlation with the progression of atherosclerotic plaque lesions[19].In a prospective cohort study involving 40 patients with ACS who underwent successful PCI, a significant correlation was observed between Lp-PLA2 concentration and atherosclerotic plaque volume,

as quantified by intravascular ultrasound (IVUS)[20].In a seminal article published in The Lancet, Zhao et al. demonstrated a significant correlation between elevated serum Lp-PLA2 concentrations and the risk of ACS[21].Consequently, the findings of this study indicate that in patients with ACS, Lp-PLA2 exacerbates the severity of coronary artery lesions through the promotion of inflammatory responses. This conclusion aligns with the perspective of Wang et al.who also demonstrated a significant correlation between serum Lp-PLA2 levels and the coronary lesion severity in CHD[22].The concentration of Lp-PLA2 serves as an independent predictive biomarker for assessing the severity of coronary artery lesions, as quantified by the Gensini score, in patients with ACS.

Furthermore, this study revealed that Lp-PLA2 levels in patients with ACS demonstrated a significant correlation with cardiovascular endpoint events following PCI.The ROC curve demonstrates that Lp-PLA2 exhibits significant predictive value for cardiovascular endpoint events following PCI in patients with ACS. The area under the curve (AUC) is 0.622, with an optimal cut-off value of 170.67, yielding a sensitivity of 55.8% and a specificity of 69.5%.Patients exhibiting elevated Lp-PLA2 concentrations in the event group demonstrated significantly higher levels compared to the non-event group. Subsequent COX regression analysis, incorporating adjustments for smoke, TC, and LDL-C, revealed that elevated Lp-PLA2 concentrations maintained a positive correlation with cardiovascular endpoint events following PCI in ACS patients (RR: 2.08).Simultaneously, this investigation revealed that the severity of coronary artery lesions demonstrated no significant correlation with the incidence of cardiovascular endpoint events in ACS patients following PCI. Consequently, we hypothesize that Lp-PLA2 may influence the occurrence of cardiovascular endpoint events through mechanisms beyond the extent of coronary artery lesions.Isabel et al. demonstrated that the concentration of Lp-PLA2 within atherosclerotic plaques exhibits a significant correlation with plaque stability[8].The elevation of Lp-PLA2 concentration within atherosclerotic plaques may induce the expansion of lipid cores, resulting in the attenuation of fibrous caps and consequently elevating the risk of plaque rupture.In the investigation conducted by Liu et al., it was demonstrated that the rupture of atherosclerotic plaques induces the upregulation of Lp-PLA2 expression, which exhibits pronounced expression in macrophages within vulnerable fibrous caps[23].Elevated concentrations of Lp-PLA2 can activate inflammatory cells to secrete substantial quantities of interleukin-6 (IL-6), consequently elevating the risk of adverse cardiovascular events[24].Empirical studies have demonstrated that low concentrations of Lp-PLA2 may contribute to the retardation of atherosclerosis progression and the reduction of cardiovascular event incidence[25].Mourouzis et al.demonstrated a significant correlation between Lp-PLA2 concentration and the clinical prognosis of ACS[26].In the systematic review conducted by Yannis et al. [27], which encompassed 14 case-control and cohort studies involving over 200,000 patients, the adjusted odds ratio for the association between Lp-PLA2 concentration and cardiovascular events was determined to be 1.60.In the meta-analysis conducted by Madjid et al., a total of 33 studies on Lp-PLA2 were included, with 30 of these studies demonstrating that elevated Lp-PLA2 concentrations were predictive of cardiovascular events[28].Consequently, the findings of this study demonstrate that in patients with ACS, Lp-PLA2 exacerbates the progression of coronary artery lesions and plaque destabilization through

inflammatory mechanisms. This process not only contributes to the onset of ACS but also induces the upregulation of Lp-PLA2 expression, thereby amplifying the inflammatory cascade and adversely impacting patient prognosis. Furthermore, Lp-PLA2 concentration serves as an independent prognostic indicator for cardiovascular endpoint events.

Finally, this study demonstrated a positive correlation between Lp-PLA2 concentration and both smoke and LDL-C. As established risk factors for CHD, their significant contributions to the pathogenesis and progression of CHD are well-documented.The substantial quantity of oxygen free radicals generated in smoke can induce peroxidation of polyunsaturated fatty acids within the cell membrane, consequently exacerbating oxidative stress[29].Previous research has demonstrated that the concentration of ox-LDL is significantly elevated in smokers[30], and ox-LDL has been shown to upregulate the expression of Lp-PLA2[31].Recent empirical studies have demonstrated that the risk of elevated serum Lp-PLA2 concentration progressively increases in overweight and obese male individuals, correlating with both the cumulative duration of smoking and the daily cigarette consumption[32].Meanwhile, LDL-C serves as the primary binding target for Lp-PLA2, accounting for approximately 80% of all lipoproteins bound to Lp-PLA2. Lp-PLA2 exhibits preferential distribution in desialylated low-density lipoprotein (dsLDL), which is particularly susceptible to oxidation. This distribution enhances the affinity of macrophages for scavenger receptor class B type I (SR-BI), thereby accelerating foam cell formation. Furthermore, this process promotes the development of atherosclerotic plaques and mediates macrophage apoptosis[33-35].The activity of LDL-Lp-PLA2 is predominantly attributable to the enzymatic activity of Lp-PLA2[36].

Research findings have demonstrated that over 50% of the therapeutic efficacy of statin medications in mitigating cardiovascular events can be attributed to the reduction of Lp-PLA2 activity[37].As proven by Jabor et al.lipid-lowering therapy lowered the serum Lp-PLA2 level while reducing the serum LDL-C level[38].

Consequently, a reciprocal and synergistic relationship exists between Lp-PLA2 and certain conventional risk factors for coronary heart disease. In the pathogenesis, progression, and prognosis of ACS, Lp-PLA2 in conjunction with traditional coronary heart disease risk factors collectively exert significant influence. Through the amplification of inflammatory processes, these factors ultimately contribute to the onset of cardiovascular events.

# 4 Study limitations

This study was conducted at a single center. Given the relatively limited sample size, potential selection bias cannot be ruled out. Furthermore, the study design involved only a single measurement of Lp-PLA2 concentration in each ACS patient post-admission, without accounting for the potential impact of dynamic fluctuations in Lp-PLA2 levels on the outcomes. Additionally, the presence of unadjusted confounding factors remains a possibility. Consequently, multicenter, large-scale observational studies are warranted to validate our findings.

# 5 Conclusion

Although Lp-PLA2 concentration is not currently recognized as a therapeutic target, its potential as an emerging biomarker for predicting the severity and prognosis of coronary artery lesions in coronary heart disease warrants significant attention. It is advisable to consider implementing a prevention and treatment strategy for coronary heart disease that integrates Lp-PLA2 concentration with traditional risk factors in the target population.A comprehensive understanding of the correlation between Lp-PLA2 levels and the severity of coronary artery lesions in ACS patients, along with its association with cardiovascular endpoint events post-PCI and traditional risk factors.It can provide evidence-based and reliable guidance for clinicians in the diagnosis, therapeutic intervention, and risk stratification of patients with ACS.

# 6 Data availability

All data generated or analyzed during this study are included in this article. Original research data cannot be made public because it may involve sensitive personal information.but are available from the corresponding author on reasonable request.

# Abbreviations

Lp-PLA2:lipoprotein-associated phospholipase A2.TIA:Transient ischemic attack.ACS:acute coronary syndrome.CHD:coronary heart disease.PCI:percutaneous coronary intervention.AS:atherosclerosis.CAG:coronary angiography.LDL-C:low density lipoprotein-cholesterol.OR:odds ratio.RR:relative risks.ROC:receiver operating characteristic.AUC:area under the curve.IVUS:intravascular ultrasound.Lyso-PC:lysophosphatidylcholine.oxFFA:oxidized free fatty acids.ox-LDL:oxidized low-density lipoprotein.dsLDL:desialylated low-density lipoprotein.BMI:body mass index.CI:confidence interval.cTNI:cardiac troponin I.

# Declarations

### Competing interests

The authors declare no competing interests.

### Ethical approval and consent to participate

The approval for this study was obtained from the Ethics Committee of the First People's Hospital of Chenzhou(Ethic2025010),Clinical trial number: not applicable. Since data were evaluated retrospectively and solely obtained for the study purposes, a requirement of informed consent was waived by the Ethics Committee of the First People's Hospital of Chenzhou. All study methods were performed in accordance with Helsinki Declaration.

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# Author Contribution

G.Zand ZH.W. conceived and designed the study. G.Z.and XW.S and WY.J collected the data. G.Zand L.H.. analyzed the data. G.Z.drafted the manuscript. XJ.and YJ.P reviewed the manuscript. All authors read and approved the final draft.

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We acknowledge the dedicated efforts of all staff involved in implementing the intervention and evaluation components of the study.

# Data Availability

All data generated or analyzed during this study are included in this article. Original research data cannot be made public because it may involve sensitive personal information.but are available from the corresponding author on reasonable request.

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# Tables

Tables 1 to 8 are available in the Supplementary Files section.

# Figures

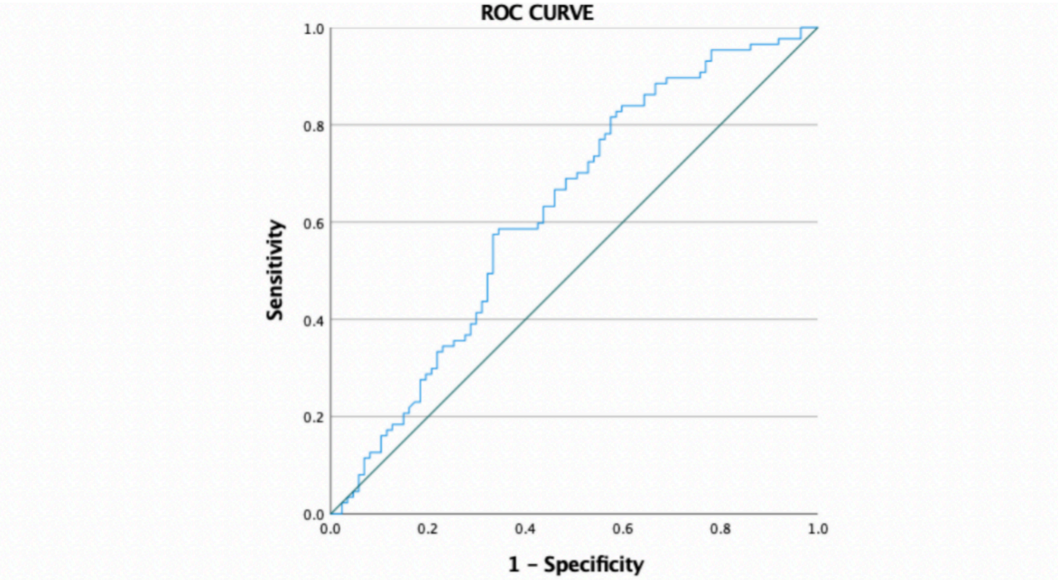



**Figure 1 The ROC curve of Lp-PLA2 demonstrates its efficacy in predicting the severity of coronary artery lesions（Gensini score）in patients with ACS**

## Figure 1

See image above for figure legend

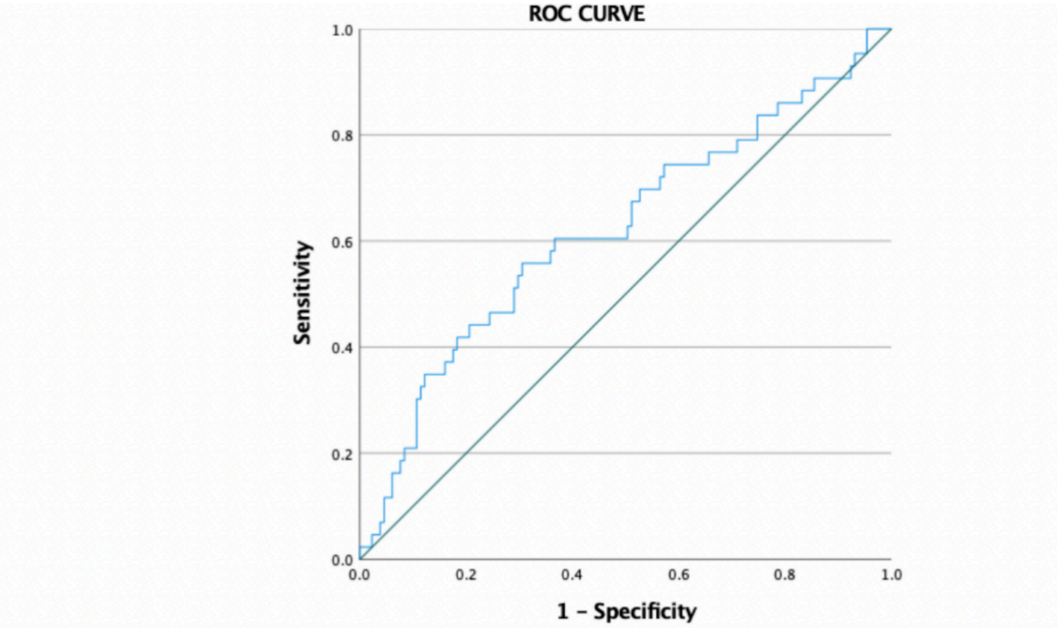



**Figure 2 The ROC curves illustrating the predictive performance of Lp-PLA2 for cardiovascular endpoint events in ACS patients following PCI**

## Figure 2

See image above for figure legend

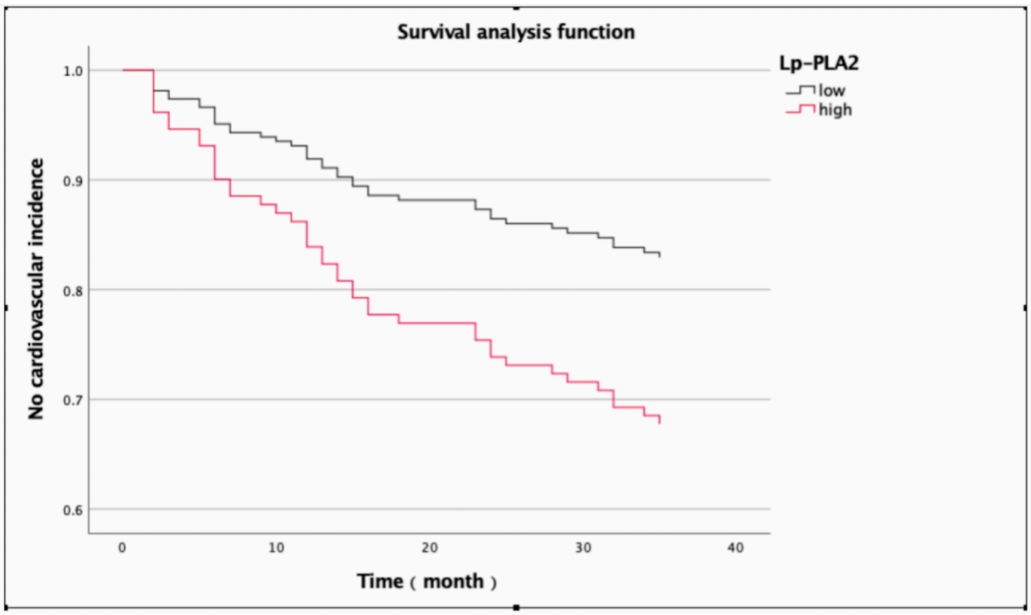



Figure 3 The Cox regression analysis function graph illustrating the relationship between Lp-PLA2 levels and cardiovascular endpoint events post-PCI in patients with ACS

### Figure 3

See image above for figure legend

## Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Table101.png
- Table201.png

- Table301.png
- Table401.png
- Table501.png
- Table601.png
- Table701.png
- Table801.png