ORIGINAL ARTICLE

Lipoprotein(a) Augments Coronary Risk Estimation in Type 2 Diabetes: A Cross-sectional Study

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Abstract

Objective: Risk estimation tools have been developed to predict coronary heart disease (CHD) in type 2 diabetes (T2D). To evaluate augmentation following the addition of lipoprotein(a) [Lp(a)] to risk calculation, we performed a pilot study.

Methods: A total of 90 successive T2D patients were included. Details of clinical and biochemical features were obtained. Lp(a) was determined using ELISA. CHD risk estimation was performed using Framingham, QRISK-3, SCORE-2D, INTERHEART, and European Atherosclerosis Society (EAS) algorithms with and without Lp(a). Descriptive statistics are reported.

Results: Mean age of patients was 55.0 ± 8 years, BP systolic/diastolic 133.7 ± 12 / 95.0 ± 9 mm Hg, body mass index (BMI) 26.0 ± 1.9 kg/m^2, waist–hip ratio 0.96 ± 0.08, fasting glucose 198.0 ± 38 mg/dL, HbA1c 9.3 ± 1.3%, total cholesterol 197.0 ± 26 mg/dL, LDL cholesterol 114.2 ± 25 mg/dL, non-HDL cholesterol 153.8 ± 27 mg/dL, and triglycerides 197.8 ± 44 mg/dL. Lp(a) was mean 23.1 ± 9.7 mg/dL and median 22.0 (25–75 IQR 15.9–29.5) mg/dL. Mean risk scores were Framingham 11.2 ± 8.7, QRISK-3 28.6 ± 15.3, INTERHEART 21.0 ± 6.0, SCORE-2D 14.9 ± 8.3, and EAS 29.2 ± 15.2. Patients with raised Lp(a) >30 mg/dL had higher levels of total, LDL, and non-HDL cholesterol and triglycerides ($p < 0.01$). Spearman's correlation of Lp(a) with risk scores was Framingham 0.127, QRISK-3 0.174, INTERHEART 0.137, SCORE-2D 0.050, and EAS 0.320, while EAS-Lp(a) was 0.397. In different risk algorithms, high risk for CHD were: Framingham 14.4%, QRISK-3 64.4%, INTERHEART 45.6%, SCORE-2D 30.0%, EAS 71.1%, and EAS with Lp(a) 74.4%. Area under the curve (AUC) for Lp(a) with various scores were Framingham 0.53 (CI: 0.39–0.68; $p = 0.644$), QRISK-3 0.57 (CI: 0.42–0.71), INTERHEART 0.55 (CI: 0.39–0.69), SCORE-2D 0.47 (CI: 0.32–0.61), EAS 0.65 (CI: 0.50–0.79), and EAS-Lp(a) 0.68 (CI: 0.54–0.83). In addition, adding Lp(a) to the EAS risk calculator increased risk reclassification by a range of 4.6–19.3%.

Conclusion: Substantial variation in coronary artery disease (CAD) risk prediction using various clinical algorithms is observed in T2D. The EAS algorithm provides the most robust estimate. The addition of Lp(a) to the risk algorithms augments risk stratification significantly. The results of this pilot study need confirmation with larger prospective studies.

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Introduction

Lipoprotein(a) [Lp(a)] has emerged as a major coronary artery disease (CAD) risk factor following persuasive data from epidemiological, case–control, and Mendelian randomization studies and clinical trials.[1,2] Studies have also reported that it is an important risk factor for premature CAD in patients with and without type 2 diabetes (T2D).[2–6] Lp(a) consists of apolipoprotein (apo) B100 covalently bound to apo(a).[7] Lp(a) characteristically inherits atherogenicity from both apoB and apo(a), as well as prothrombogenic and proinflammatory traits from apo(a). A major comorbidity of diabetes is CAD and is estimated to affect more than a third to half of all patients with diabetes.[8,9] Many risk scoring algorithms have been developed to predict CAD in T2D, including the Framingham risk score, QRISK-3 from the UK, SCORE of the European Society of Cardiology (SCORE-2D), INTERHEART risk score, and European Atherosclerosis Society (EAS) risk score.[10] Previous studies have reported that most perform suboptimally in non-European populations in general and T2D in particular.[10–13]

Lp(a) and diabetes are both established risk factors for the development of CAD.[13] However, studies trying to link both risk factors find an inverse association between Lp(a) and risk of prevalent and incident diabetes.[4] It is not clear whether this association is causal or whether it is due to Lp(a) itself, the length of the apo(a) isoforms, or both. The results of Mendelian randomization studies are highly heterogeneous.[1,14] Only part of the observed association of Lp(a) with diabetes can be explained by causality. It may also be due to reverse causation, comorbidities, or medications. Previous studies have reported that patients with T2D do not exhibit higher levels of Lp(a).[13] Results from the Biomarker for Cardiovascular Risk Assessment across Europe (BiomarCaRE) consortium indicated that elevated Lp(a) was robustly associated with an increased risk for CAD in individuals with T2D.[14] Therefore, the present study was undertaken to assess (1) the correlation of CAD risk scores with Lp(a) in patients with T2D, and (2) additional CAD risk prediction with Lp(a) using the EAS risk prediction algorithm.

Methods

This hospital-based cross-sectional study was conducted on 90 successive patients (men 63, women 27) with T2D presenting to the medical outpatient department. The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from all the enrolled individuals. The inclusion criteria were T2D, age 40–70 years, without microvascular or macrovascular complications of diabetes. T2D was diagnosed using the criteria used in recent Indian studies.[15] Participants were excluded if they had a previous history of cardiovascular disease, macrovascular complications of diabetes, were terminally ill, had severe liver or renal insufficiency, type 1 diabetes, cancer, thyroid dysfunction, severe mental illness, pregnancy, peripheral artery disease, hemolytic disease, severe disabilities, or were using drugs interfering with Lp(a) metabolism such as niacin and chronic use of steroids. A detailed questionnaire was obtained for participant information, including demographic characteristics, clinical features, diabetes duration, use of medications, and smoking status. Anthropometric measurements were recorded using standard procedures. Each participant's height and weight were measured, and body mass index (BMI) was calculated by

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dividing the weight in kilograms by height in meters squared. Waist circumference and hip circumference were measured, and the waist–hip ratio was calculated. Blood pressure was measured by a mercury sphygmomanometer. Hypertension was defined as systolic blood pressure >140 mm Hg and/or diastolic blood pressure >90 mm Hg or current use of any treatment with antihypertensive medications.

Blood samples were collected after an overnight fast into appropriate Vacutainer tubes. The serum or plasma was removed within an hour and refrigerated at −80°C for analysis of Lp(a). Measurement of serum glucose, hemoglobin A1C (HbA1C), total cholesterol (TC), triglyceride (TG), and HDL-C was conducted on fresh samples using standard procedures with an automated analyzer (Beckman AU800) and enzymatic assays. Low-density lipoprotein cholesterol (LDL-C) levels were calculated using the Friedewald formula, except when triglyceride levels were >400 mg/dL. Lp(a) was estimated using a sandwich enzyme-linked immunosorbent assay kit. External validation was performed for Lp(a) estimation, with intra- and inter-assay coefficients of variation of 4.5 and 6.7%, respectively.

Statistical Analyses

This is an observational pilot study, and the study sample size was determined using previously available studies from India. Cardiovascular risk estimation was performed using multiple algorithms—Framingham risk score (FRS),[16] QRISK-3 (UK),[17] SCORE-2 Diabetes (SCORE-2D) of the European Society of Cardiology,[18] INTERHEART risk score,[19] and European Atherosclerosis Society (EAS) score.[20] All the risk scores were calculated using online calculators.[21] Mean values of risk scores were determined. Participants were stratified into 3 categories according to 10-year coronary heart disease (CHD) risk as low risk (<10%), intermediate risk (10–20%), and high risk (>20%). The Lp(a) distribution was skewed and nonnormal; hence, Spearman's correlation of individual risk scores with serum Lp(a) levels was performed to estimate the variation in risk prediction. Receiver operating curves (ROCs) were plotted for various risk scores with Lp(a) >30 mg/dL, and the area under the curve (AUC) was determined using the SPSS statistical package (version 22.0). Additional discriminant value of Lp(a) was calculated using the EAS risk scoring algorithm with and without Lp(a). The article follows the STROBE guidelines for observational studies. The STROBE checklist is attached as Supplementary File.

Results

The key characteristics of the study cohort are in Table 1. The mean age of the participants was 55.0 ± 8 years, the majority being men. A high burden of cardiovascular risk factors,

Table 1: Clinical and biochemical characteristics of the study cohort

Variable	*Total*	*Lp(a) <30 mg/dL*	*LP(a) >30 mg/dL*	*p-value*
Numbers	90	69	21	
Men	63 (70.0)	48 (69.6)	15 (71.4)	0.870
Age years	54.7 ± 8.0	54.6 ± 8.0	54.8 ± 8.1	0.905
Age-groups				
<40 years	30 (33.3)	22 (31.9)	8 (38.1)	0.712
40–59 years	34 (37.8)	27 (39.1)	7 (33.3)	0.744
60+	26 (28.9	20 (29.0)	6 (28.6)	0.998
Family history of CAD	37 (41.1)	27 (39.1)	10 (47.6)	0.489
Tobacco use (smoking/smokeless)	46 (51.1)	38 (55.1)	8 (38.1)	0.173
Alcohol use	38 (42.2)	31 (44.9)	7 (33.3)	0.346
Hypertension	47 (52.2)	36 (52.2)	11 (52.4)	0.987
Systolic BP	133.7 ± 12.0	133.6 ± 12.2	134.0 ± 11.3	0.900
Diastolic BP	95.0 ± 8.6	94.7 ± 8.6	95.8 ± 8.9	0.627
Waist circumference	94.9 ± 7.4	95.0 ± 7.6	94.7 ± 6.8	0.892
Waist–hip ratio	0.96 ± 0.08	0.96 ± 0.08	0.96 ± 0.08	0.921
BMI	26.0 ± 1.9	26.1 ± 2.2	25.9 ± 1.8	0.680
Diabetes duration	7.3 ± 5.4	7.1 ± 5.3	8.2 ± 5.9	0.406
Biochemical parameters				
HbA1c (mean)	9.3 ± 1.2	9.4 ± 1.2	9.0 ± 1.3	0.217
HbA1c >7.0%	90 (100)	69 (100.0)	21 (100.0)	1.00
Total cholesterol, mean	197.0 ± 26.4	191.6 ± 24.2	214.6 ± 26.1	<0.001
High cholesterol >200 mg/dL	35 (38.9)	19 (27.5)	16 (76.2)	<0.001
LDL cholesterol, mean	114.2 ± 24.7	109.7 ± 22.1	130.1 ± 26.3	0.001
High LDL cholesterol >100 mg/dL	68 (75.6)	49 (71.0)	19 (90.5)	0.069
Non-HDL cholesterol	153.8 ± 26.6	147.3 ± 23.9	175.1 ± 24.0	<0.001
Non-HDL cholesterol >130 mg/dL	73 (81.1)	53 (76.8)	20 (95.2)	0.059
Triglycerides	197.8 ± 44.4	189.5 ± 42.7	225.0 ± 39.5	0.001
High triglycerides >150 mg/dL	82 (91.1)	61 (88.4)	21 (100.0)	0.102
LDL:HDL ratio	2.7 ± 0.89	2.6 ± 0.80	3.4 ± 0.87	<0.001
Triglyceride:HDL ratio	4.8 ± 1.6	4.4 ± 1.3	5.9 ± 1.8	<0.001
Lipoprotein(a) mean	23.0 ± 9.7	19.0 ± 6.2	36.3 ± 6.9	<0.001
Lipoprotein(a) median, IQR	22.0 (15.9–29.5)	22.0 (14.6–24.2)	33.8 (32.1–39.4)	<0.001

family history, hypertension, smoking, or tobacco use is observed. The mean systolic/diastolic BP was 133.7 ± 12/95.0 ± 9 mm Hg, BMI 26.0 ± 1.9 kg/m^2, and waist–hip ratio 0.96 ± 0.08. Biochemical analyses showed mean fasting glucose 198.0 ± 38 mg/dL, HbA1c 9.3 ± 1.3%, total cholesterol 197.0 ± 26 mg/dL, LDL cholesterol 114.2 ± 25 mg/dL, non-HDL cholesterol 153.8 ± 27 mg/dL, and triglycerides 197.8 ± 44 mg/dL. A significant proportion of participants had raised levels of total cholesterol (>200 mg/dL, 38.9%), LDL cholesterol (>100 mg/dL, 75.6%), non-HDL cholesterol (>130 mg/dL, 81.1%), and triglycerides (>150 mg/dL, 91.1%). The mean level of Lp(a) was 23.0 mg/dL, with a median value of 22.0 mg/dL (25–75% IQR 15.9–29.5 mg/dL). A total of 21 patients (23.3%) had elevated Lp(a) levels of >30 mg/dL. Clinical and biochemical characteristics of participants with Lp(a) <30 mg/dL (n = 69) were compared with those of participants with raised Lp(a) >30 mg/dL (n = 21). Clinical characteristics and risk factors are similar (Table 1). However, a subgroup of participants exhibiting elevated levels of Lp(a) consistently demonstrates increased levels of total cholesterol, LDL-C, non-HDL-C, and triglycerides (p < 0.05 for all).

Table 2 shows the mean 10-year CHD risk prediction scores using various risk assessment tools. Mean risk scores were the highest for EAS (29.2 ± 15.2) and QRISK-3 (28.6 ± 15.3) risk calculators compared to others. Increased CHD risk (>10%, 10-year risk) using various risk calculators was for Framingham 14.4% (n = 13), QRISK-3 64.4% (n = 58), INTERHEART 45.6% (n = 41), SCORE-2D 30.0% (n = 27), and EAS 71.1% (n = 64). Addition of Lp(a) to the EAS risk calculator enhanced the CHD risk to 74.4% (+4.6%) (n = 67). Correlation between individual CHD risk prediction scores and Lp(a) levels using Spearman's correlation coefficient (rho), as well as parametric (Pearson's r), is in Table 3. Weak correlation is observed between Lp(a) levels and Framingham (p = 0.234), INTERHEART (p = 0.197), and SCORE-2D (p = 0.641) risk scores; intermediate for QRISK-3 risk score (p = 0.100); and significant for EAS risk prediction score (rho = 0.320, p = 0.002). The addition of Lp(a) to the EAS risk score further enhances the correlation (rho = 0.397, p < 0.001).

Receiver operating curve (ROC) analyses for risk prediction using various risk scores and Lp(a) levels of >30 mg/dL are in Figure 1. Mean area under the curve (AUC) with various scores were for Framingham 0.53 (CI: 0.39–0.68, p = 0.644), QRISK-3 0.57 (CI: 0.42–0.71, p = 0.347), INTERHEART 0.55 (CI: 0.39–0.69, p = 0.520), SCORE-2D 0.47 (CI: 0.32–0.61, p = 0.654), EAS 0.65 (CI: 0.50–0.79, p = 0.040), and EAS calculator with Lp(a) (>30.0 mg/dL) 0.68 (CI: 0.54–0.83, p = 0.011). The ROC analysis highlights the enhanced risk discrimination of +4.6% when Lp(a) levels were incorporated into the EAS risk calculator and up to 19.6% with other risk estimation algorithms.

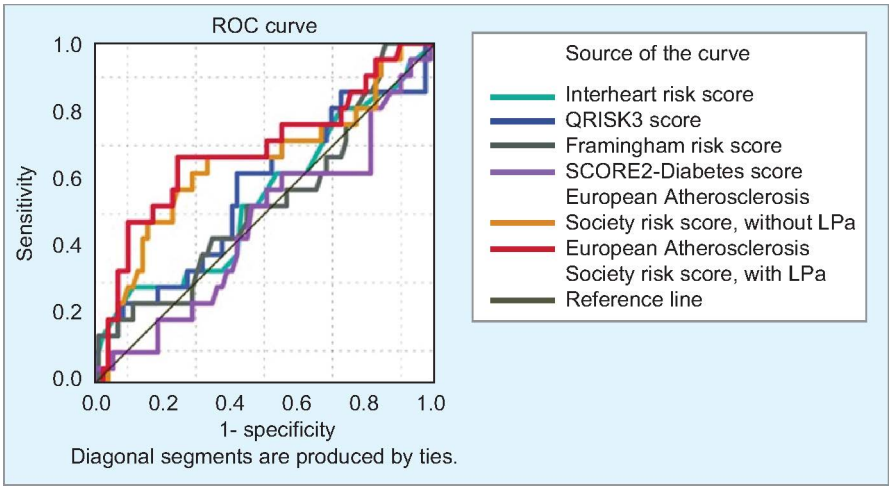

Fig. 1: Area-under-the-curve analyses for association of Lp(a) levels (>30 mg/dL) with cardiovascular risk scores among T2D patients using various algorithms. The highest association is with the European Atherosclerosis Society (EAS) risk score incorporating Lp(a)

Discussion

Coronary heart disease is the most important cause of deaths in T2D.[8] The present study shows that various commonly available risk prediction tools perform suboptimally for CHD risk stratification in diabetes. We also show that the addition of lipoprotein(a) in various risk calculators enhances risk stratification. Recent studies have highlighted the importance of Lp(a) as a coronary risk factor in T2D,[22,23] and the present study, though small, suggests that Lp(a) should be routinely assessed in T2D to estimate CAD risk.

The American College of Endocrinology Consensus (2020) considers T2D as a CHD risk equivalent, and all individuals with it are classified as very high risk.[24] The Framingham risk calculator,[16] American College of Cardiology/American Heart Association (ACC/AHA),[25] and American Diabetes Association (ADA)[26] guidelines have incorporated T2D in risk calculations and do not recommend additional measures for risk estimation. The European Society of Cardiology (ESC) recommends estimation of CAD risk using a diabetes-specific SCORE-2D risk calculator.[18] Our study shows (Table 2) that Framingham and European SCORE-2D risk scores classify only a quarter to half of T2D patients as high risk, highlighting the limitations of the applicability of these risk scores for our patients. Studies have reported that raised Lp(a) levels are associated with a 2–3-fold higher risk.[22,23] In the present study, when Lp(a) is added to the usual risk calculators, there is a 5–20% greater discrimination. The ROC analysis highlights the enhanced risk discrimination when Lp(a) levels were incorporated into the EAS risk algorithm. Figure 1 also demonstrates a sensitivity of the EAS score (with or without Lp(a) inclusion) at 75% even at a specificity of

Table 2: Coronary risk scores for the study cohort with high, intermediate, and low cardiovascular risk

Risk score	*Mean ± SD*	*High risk*	*Intermediate*	*Low risk*
Framingham risk score	11.2 ± 8.7	13 (14.4)	35 (38.9)	42 (46.7)
QRISK-3 risk score	28.6 ± 15.3	58 (64.4)	22 (24.4)	10 (11.1)
INTERHEART risk score	21.0 ± 6.0	41 (45.6)	44 (48.9)	5 (5.6)
SCORE2-diabetes score	14.9 ± 8.3	27 (30.0)	46 (51.1)	17 (18.9)
EAS risk score	29.2 ± 15.2	64 (71.1)	18 (20.0)	8 (8.9)
EAS risk score with Lp(a)	31.8 ± 16.5	67 (74.4)	17 (18.9)	6 (6.7)

EAS European Atherosclerosis Society; INTERHEART Study; QRISK QRESEARCH cardiovascular risk; SCORE2-D Systematic Coronary Risk Evaluation-2 Diabetes

Table 3: Nonparametric (Spearman) and parametric (Pearson) correlation of Lp(a) with various risk scores

	Spearman's rho	*Pearson's r*
Framingham risk score	0.127 (0.234)	0.080 (0.446)
QRISK-3 risk score	0.174 (0.100)	0.136 (0.202)
INTERHEART risk score	0.137 (0.197)	0.073 (0.495)
SCORE2-diabetes	0.050 (0.641)	0.021 (0.845)
EAS risk score	0.320 (0.002)	0.354 (0.001)
EAS risk score with Lp(a)	0.397 (<0.001)	0.418 (<0.001)

EAS European Atherosclerosis Society; INTERHEART Study; QRISK-3 cardiovascular risk; SCORE2-D Systematic Coronary Risk Evaluation-2 Diabetes

10–60%. This observation suggests that the addition of Lp(a) to the EAS risk calculator would allow clinicians to identify higher risk in T2D patients earlier and more robustly. Beyond the presentation of statistics, the data indicate that risk reclassification increased by a range of 4.6–19.3%, contingent upon the specific model employed.

Data from the UK Biobank participants (n = 4,60,506) show that the risk of myocardial infarction increases linearly beyond Lp(a) >30 mg/dL and peaks at >150 mg/dL.[27] However, there is no international consensus for the incorporation of Lp(a) in risk assessment scores; the EAS is an exception. The addition of Lp(a) in the risk assessment tool, as done by the EAS, is a step forward. Our study shows that other risk assessment tools should also do likewise. Other risk prediction tools, such as the coronary artery calcium score and raised hsCRP (highly sensitive C-reactive protein), can influence the decision for intensive lipid modifications in T2D patients.[10] It is suggested that a more robust risk assessment tool should be developed for the identification of CAD risk in T2D. Currently, there are no approved medications for lowering Lp(a), although many monoclonal antibodies and small interfering RNA (siRNA) molecules are in the pipeline.[28,29] Presently, guidelines suggest that individuals with raised Lp(a) should be advised to use high-intensity statins to control apolipoprotein B and PCSK9 inhibitors that can reduce Lp(a) levels by 25–30%.[30]

The study has several limitations. In addition to those previously mentioned, these include a small sample size, which limits the robustness of the findings and reduces confidence in the conclusions. Furthermore, the study's single-center design may not adequately represent the broader population of patients with T2D, and its hospital-based cohort design may introduce selection bias and limit generalizability. The use of a sandwich enzyme-linked immunosorbent assay, rather than more robust methods for measuring Lp(a) particle numbers and size, is another limitation. Additionally, the cross-sectional design of the study precludes the establishment of causality and the ability to track changes over time. There are no large epidemiological studies that have measured Lp(a) in the Indian population, and our findings are consistent with smaller studies that report raised Lp(a) in a quarter of the population.[31] However, the median values are lower than those reported from emigrant South Asians.[32,33] Larger studies among diabetics and non-diabetics are required to accurately identify the burden of raised Lp(a) in our country. Due to the absence of long-term follow-up, this study is unable to evaluate potential changes in lipoprotein(a) levels over time or their association with future cardiovascular outcomes. The current findings do not warrant immediate modifications to guidelines or assert causal relationships. Prospective studies or Mendelian randomization studies are needed to definitively identify the role of this lipoprotein in the pathogenesis of CAD in South Asians, a population with one of the highest rates of CAD in the world.[9]

In the present study, the rationale for incorporating Lp(a) is grounded in evidence and characterized by a measured approach. Nevertheless, the establishment of definitive clinical practice requires broader participant enrollment and extended follow-up periods.

Conclusion

Raised Lp(a) is an important cardiovascular risk factor. The study shows that raised Lp(a) is present in a quarter of patients with T2D. Uniquely, the present study shows that incorporating Lp(a) values into multiple risk scoring algorithms significantly augments the risk and consistently demonstrates enhanced discriminatory power. Larger and prospective cohort studies are required to confirm these findings in our population.

Ethical Consideration

Institutional ethics committee approval was obtained before the study; therefore, the study was performed according to the ethical standards of the 2013 Declaration of Helsinki. Written informed consent was obtained from each study participant before inclusion. All information collected was kept confidential.

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