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Estimation of Serum Lipoprotein(a) and Uric Acid in Type 2 Diabetes Mellitus - A Case Control Study

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is associated with increased cardiovascular risk that is not fully explained by conventional risk factors. Emerging biomarkers such as lipoprotein(a) [Lp(a)] and serum uric acid may contribute to this residual risk.
Objectives: To evaluate and compare serum Lp(a) and uric acid levels in patients with T2DM and healthy controls, and to assess their association with conventional lipid parameters.
Methods: This case–control study included 80 participants (40 T2DM patients and 40 age and sex matched healthy controls) conducted at a tertiary care centre in South India. Fasting blood samples were analysed for Lp(a), uric acid, glucose, HbA1c, and lipid profile using standardised methods. Statistical analysis was performed using Student's t-test, with $p < 0.01$ considered significant.
Results: Serum Lp(a) and uric acid levels were significantly higher in T2DM subjects compared to controls ($p < 0.01$). Diabetic subjects also showed significantly elevated total cholesterol, triglycerides, LDL-C, VLDL-C, and reduced HDL-C levels. Glycaemic parameters were significantly higher in the T2DM group.
Conclusion: Elevated Lp(a) and uric acid levels in T2DM patients may contribute to increased cardiovascular risk beyond traditional lipid parameters. Their inclusion in routine evaluation may improve risk stratification.

Key Words: Type 2 Diabetes Mellitus, Lipoprotein(a), Serum Uric Acid, Diabetic Dyslipidaemia, Biomarkers, Hyperuricemia, Atherosclerosis, Cardiometabolic Risk

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is one of the most prevalent chronic metabolic conditions worldwide, contributing significantly to the global burden of non-communicable diseases. Characterised by insulin resistance, relative insulin deficiency, and chronic hyperglycaemia, T2DM is associated with a wide spectrum of microvascular and macrovascular complications. Among these, cardiovascular disease (CVD) remains the leading cause of morbidity and mortality. Individuals with diabetes have a two to four fold higher risk of myocardial infarction, stroke, coronary artery disease, and peripheral arterial disease compared with the general population (1).
Although glycaemic dysregulation plays an important role in vascular complications, the elevated cardiovascular risk in T2DM cannot be attributed solely to hyperglycaemia. A

range of metabolic disturbances including dyslipidaemia, oxidative stress, inflammatory activation, endothelial dysfunction, increased platelet adhesiveness, and abnormalities in coagulation and fibrinolysis contribute to accelerated atherosclerosis. Classical diabetic dyslipidaemia includes elevated triglycerides, low HDL-C and smaller, denser LDL-C particles (2). These abnormalities collectively increase atherogenic potential; however, epidemiological observations indicate that optimisation of LDL-C with statins does not fully normalise cardiovascular risk. Many patients with diabetes experience cardiovascular events even when meeting guideline-directed lipid targets, suggesting the presence of "residual cardiovascular risk" (3).

In recent years, attention has shifted to non-traditional biomarkers that may provide additional insights into CVD risk beyond conventional lipid parameters. Among these, lipoprotein(a) [Lp(a)] has emerged as an important risk determinant. Lp(a) is a unique lipoprotein particle consisting of an LDL-like core linked to apolipoprotein(a) via a disulfide bond; the apo(a) component structurally resembles plasminogen, enabling Lp(a) to competitively inhibit fibrinolysis. This dual atherogenic and thrombogenic property makes Lp(a) a potent mediator of vascular risk (4). Multiple large prospective studies and Mendelian randomisation analyses have confirmed Lp(a) as a causal risk factor for atherosclerotic CVD irrespective of LDL-C concentration (5).

Unlike other lipoproteins, Lp(a) levels are predominantly genetically determined, with minimal influence from lifestyle or environmental factors. Therefore, individuals with inherently high Lp(a) levels may remain at elevated cardiovascular risk even with aggressive statin therapy, as statins do not lower Lp(a) and may in some cases slightly increase it (6). Emerging therapeutic approaches, including antisense oligonucleotides, have shown promise in lowering Lp(a), but these treatments are not yet widely available.

Serum uric acid, an end product of purine metabolism has historically been viewed primarily in the context of gout. However, accumulating evidence suggests that hyperuricaemia is associated with cardiometabolic abnormalities including hypertension, renal dysfunction, endothelial injury, oxidative stress, and systemic inflammation (7). In T2DM, elevated uric acid levels may result from insulin resistance, which reduces renal urate excretion. Several epidemiological studies have demonstrated strong associations between higher uric acid levels and increased risk of cardiovascular morbidity and all-cause mortality in diabetic populations (8, 9). Given these observations, evaluating Lp(a) and uric acid concentrations in individuals with T2DM may provide clinically meaningful information regarding additional mechanisms contributing to their heightened cardiovascular risk. Although several international studies have examined these biomarkers in the context of diabetes, data from South Asian populations remain limited. South Asians have a disproportionately high cardiometabolic risk, making it essential to understand the behaviour of these biomarkers in such populations.

The present study aims to evaluate and compare serum Lp(a) and uric acid levels in T2DM and age-matched healthy controls using standardised methods. It also explores the association of these biomarkers with traditional lipid parameters to assess their potential contribution to cardiovascular risk stratification in diabetic individuals. Understanding the interplay between Lp(a), uric acid, and conventional risk factors may help refine assessment strategies and identify individuals at higher risk who may benefit from more intensive interventions.

MATERIALS AND METHODS

Study Design and Setting

A case–control study was conducted over an 18-month period at a tertiary care teaching

hospital in South India. The study compared serum lipoprotein(a) [Lp(a)] and serum uric acid levels in individuals with diagnosed type 2 diabetes mellitus (T2DM) and age- and sex-matched healthy controls.

Study Population

A total of 80 participants were included, comprising 40 patients with T2DM (cases) and 40 apparently healthy, non-diabetic individuals (controls). Participants in both groups were aged between 40 and 60 years.

Inclusion criteria:

Individuals aged 40–60 years diagnosed with T2DM according to the American Diabetes Association 2018 criteria (fasting plasma glucose ≥126 mg/dL, postprandial plasma glucose ≥200 mg/dL, and/or HbA1c ≥6.5%) were included as cases. Age- and sex-matched apparently healthy individuals without diabetes were recruited as controls. Written informed consent was obtained from all participants.

Exclusion criteria:

Individuals with type 1 diabetes mellitus; a history of cardiovascular, hepatic, neurological, respiratory, endocrine, infectious, malignant, or haematological disorders; alcohol use or smoking; severe immunodeficiency; pregnancy or gestational diabetes; hormone replacement therapy; or those receiving statins, insulin, thiazide diuretics, uric acid–lowering drugs, or multivitamin supplements were excluded from the study.

Ethical Approval

The study protocol was reviewed and approved by the Institutional Ethics and Research Committee (BMC/PG/124/2018-19), and the study was conducted in accordance with the ethical standards laid down in the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment.

Sample Collection

Following an overnight fast, approximately 6 mL of venous blood was collected from each participant under aseptic conditions. Blood samples were divided into two portions: one collected in an EDTA tube for glycated haemoglobin estimation and the other collected in a plain tube for serum separation. Relevant clinical history and findings from physical examination were recorded using a structured proforma.

Laboratory Methods and Specimen Handling

Venous blood (6 mL) was collected under aseptic conditions after overnight fasting. Whole blood was collected in EDTA tubes for glycated haemoglobin estimation, and serum was separated from clotted blood by centrifugation for biochemical analyses. Serum samples were analysed immediately or stored at 2–8 °C and processed within 24 hours.

Lipoprotein(a)

Serum lipoprotein(a) [Lp(a)] was measured by latex-enhanced immunonephelometry using a commercially available reagent kit on a semi-automated protein analyzer (MISPA-i2; Agappe Diagnostics Ltd., Kerala, India). The assay was linear up to 100 mg/dL, with a lower detection limit of 1 mg/dL. The reference range applied was ≤30 mg/dL. Intra-assay coefficients of variation ranged from 1.87% to 3.77%, and inter-assay coefficients of variation ranged from 1.62% to 2.75% across control levels. Accuracy was verified using commercial lipid control materials supplied by the manufacturer.

Serum Uric Acid

Serum uric acid was estimated by an enzymatic colorimetric uricase–peroxidase method using commercial reagents on the Roche Cobas 6000 analyzer (c501 module; Roche Diagnostics, Mannheim, Germany). Absorbance was measured photometrically at 546 nm. The reference ranges applied were 3.4–7.0 mg/dL for males and 2.4–5.7 mg/dL for females.

Plasma Glucose

Fasting and postprandial plasma glucose concentrations were measured using the hexokinase enzymatic reference method on the Roche Cobas 6000 analyzer (c501 module; Roche Diagnostics, Mannheim, Germany). Results were expressed in mg/dL, with a fasting value of less than 100 mg/dL.

Glycated Haemoglobin (HbA1c)

Whole blood HbA1c was measured using ion-exchange high-performance liquid chromatography (HPLC) on the Bio-Rad D-10 system (Bio-Rad Laboratories, Hercules, CA, USA). HbA1c values were reported as percentages, with values ≤5.7% considered normal.

Lipid Profile

Serum total cholesterol was measured by the cholesterol oxidase–peroxidase (CHOD-PAP) method, triglycerides by the glycerol phosphate oxidase–peroxidase method, and HDL-cholesterol by a homogeneous enzymatic colorimetric PEG-CHOD method using commercial reagent kits on the Roche Cobas 6000 analyzer (c501 module; Roche Diagnostics, Mannheim, Germany). LDL-cholesterol was calculated using the Friedewald equation for samples with triglyceride concentrations <400 mg/dL. Reference ranges were based on National Cholesterol Education Program (NCEP) guidelines.

Statistical Analysis

Data were analysed using the Statistical Package for the Social Sciences (SPSS), version 17.0. Continuous variables were expressed as mean ± standard deviation. Normality of data distribution was assessed prior to analysis. Differences between cases and controls were evaluated using Student's t-test. A p value <0.01 was considered statistically significant.

Use of Artificial Intelligence Tools

Artificial intelligence–assisted language tools were used for language refinement and structural editing of the manuscript. The authors take full responsibility for the accuracy, integrity, and scientific content of the work.

RESULTS

Table 1. Serum Lipoprotein(a) Levels in Study Groups

Group	n	Mean (mg/dL)	SD	p value
T2DM	40	45.19	12.64	<0.01
Controls	40	10.37	5.71	

Values are expressed as mean ± SD.

Table 2. Serum Uric Acid Levels in Study Groups

Group	n	Mean (mg/dL)	SD	p value
T2DM	40	5.60	1.37	<0.01
Controls	40	3.08	0.84	

Values are expressed as mean ± SD.

Lipoprotein(a)

The mean serum lipoprotein(a) [Lp(a)] concentration was significantly higher in subjects with type 2 diabetes mellitus compared with healthy controls (45.19 ± 12.64 mg/dL vs 10.37 ± 5.71 mg/dL, p <0.01) (Table 1).

A higher proportion of diabetic subjects had serum Lp(a) levels exceeding 30 mg/dL

Serum Uric Acid

Mean serum uric acid levels were significantly higher in the T2DM group compared with controls (5.60 ± 1.37 mg/dL vs 3.08 ± 0.84 mg/dL, p < 0.01) (Table 2).

Male participants with T2DM demonstrated higher uric acid levels compared with female participants.

Glycaemic and Lipid Parameters

Subjects with T2DM exhibited significantly higher fasting plasma glucose, postprandial plasma glucose, and glycated haemoglobin levels compared with controls ($p < 0.01$). Total cholesterol, triglycerides, LDL-cholesterol, and VLDL-cholesterol levels were significantly elevated, while HDL-cholesterol levels were significantly lower in diabetic subjects compared with controls ($p < 0.01$).

DISCUSSION

The present study demonstrates that adults with T2DM exhibit significantly higher serum Lp(a) and significantly increased uric acid levels compared with age-matched healthy controls. These findings are consistent with multiple international studies and highlight the importance of evaluating non-traditional biomarkers in assessing cardiovascular risk in T2DM.

Role of Lp(a) in cardiovascular risk

Several landmark studies have shown that Lp(a) is an independent and causal risk factor for atherosclerotic cardiovascular disease. Nordestgaard et al. established its association with coronary artery disease and recommended clinical cut-off values for risk stratification [10]. Vinci et al. detailed the proatherogenic and prothrombotic mechanisms of Lp(a) and proposed its inclusion in routine risk evaluation [11]. In diabetic populations, Lim et al. demonstrated that elevated Lp(a) is associated with cardiovascular events independent of glycemic control [12].

Mechanistically, Lp(a) accelerates atherosclerosis through:

1. Oxidised phospholipid carriage, leading to endothelial inflammation [13].
2. Interference with fibrinolysis, as apo(a) competes with plasminogen [14].
3. Promotion of smooth muscle proliferation, enhancing plaque stability but also thrombosis.

In the present study, significantly elevated Lp(a) in T2DM compared with controls supports earlier findings by Gudbjartsson DF et al., who reported elevated Lp(a) in different diabetic cohorts [15]. These results highlight that Lp(a) may contribute to the residual risk seen in diabetic individuals even after LDL-C optimisation.

Inverse and complex associations

Genetic studies by Gudbjartsson et al. revealed that while Lp(a) is strongly linked to CVD, it is not causally related to T2DM incidence itself [15]. Mansson et al. further suggested that while Lp(a) influences fibrinolysis, it may not directly correlate with glycemic indices [16]. These observations, however, do not negate the role of elevated Lp(a) as an enhancer of cardiovascular risk in established diabetes.

Uric acid as a cardiometabolic marker

Hyperuricaemia is increasingly recognised as an important cardiovascular risk indicator. Feig et al. described the role of uric acid in endothelial dysfunction, oxidative stress, and renal microvascular injury [17]. Kuwabara demonstrated that elevated uric acid predicts hypertension and metabolic syndrome [18]. Kim HK et al. highlighted that high serum uric acid variability independently increases the risk of new-onset symptomatic cardiovascular disease requiring PCI in patients with type 2 diabetes. [8].

High uric acid levels:

- Reduce nitric oxide availability
- Increase oxidative stress
- Promote inflammation via NLRP3 inflammasome activation
- Activate the renin–angiotensin system

All these mechanisms amplify vascular injury in diabetic subjects. Several studies, including those by Kim et al. and Saito et al., support uric acid's independent association with cardiovascular outcomes [19,20]. Chen et al. introduced novel indices such as uric-acid-albumin ratios that predict mortality risk [21]. In the present study, significantly higher uric acid in T2DM corroborates these observations.

Clustering of cardiometabolic risk factors

The combined elevation of:

- Serum Lp(a)
- Uric acid
- LDL-C
- Triglycerides
- Fasting glucose
- HbA1c

creates a high-risk cardiometabolic phenotype typical among South Asians.

Previous studies have emphasised the value of evaluating multiple cardiometabolic biomarkers together for risk stratification, irrespective of differences in biomarker inclusion [22,23].

Clinical relevance

Given the strong evidence linking Lp(a) and uric acid with CVD:

- A one-time Lp(a) measurement is recommended for all high-risk adults, including T2DM patients.
- Uric acid monitoring may identify individuals more susceptible to endothelial dysfunction and hypertension.
- Elevated Lp(a) may justify intensified LDL-C lowering strategies, including combination therapy.
- Lifestyle measures such as weight reduction and dietary modification may reduce uric acid.

Limitations

The study has limitations:

- modest sample size
- absence of long-term cardiovascular outcome data
- lack of multivariate analysis

Nevertheless, the findings align strongly with international data.

CONCLUSION

Serum Lp(a) and uric acid levels are significantly elevated in individuals with T2DM compared with healthy controls. These biomarkers, together with classical lipid abnormalities, may contribute substantially to increased cardiovascular risk in T2DM. Inclusion of Lp(a) and uric acid assessment in routine metabolic evaluation may enhance early cardiovascular risk identification. Larger multicentric studies are needed to validate these findings and guide future therapeutic interventions.

Declaration by Authors

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