Journal of Pioneering Medical Sciences
Received: March 17, 2025 | Accepted: April 20, 2025 | Published: August 05, 2025
Volume 14, Issue 07, Pages 56-61

DOI https://doi.org/10.47310/jpms2025140710

Association of Lipid Profile with Diabetic Kidney Disease in Type 2 Diabetes Mellitus

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Abstract **Background:** Diabetes Mellitus (DM) is a common metabolic disorder resulting from insufficient insulin secretion or insulin action that leads to elevated blood glucose levels. The metabolic disorders associated with DM cause secondary pathophysiological changes in multiple organ systems leading to acute and chronic complications. Insulin resistance is frequently higher in patients with diabetes or microalbuminuria in the context of renal disease, which may accelerate the progression of Diabetic Kidney Disease. **Methods:** A cross-sectional study conducted at the Galiawa Teaching Center in Erbil, Iraq. The study included 100 patients who had been diagnosed with type 2 diabetes mellitus. lipid profile, glycated hemoglobin (HbA1C) and albumin creatinine ratio (ACR) were done for them, then statistically analyzed. **Results:** 100 participants were included, the mean age was 57±9 years, ranging from 34 to 80 years. About 60% of the participants were females and 40% were males. The most frequent category was stage A2, observed in 37 patients (37%). Additionally, 86% of participants had an eGFR within stages 1 to 3a. low-density lipoprotein (LDL) levels showed a statistically significant difference between ACR groups A1, A2 and A3 (p-value = 0.000), with the highest mean observed in group A3 (136±26 mg/dL). **Conclusion:** There is a significant correlation between mean LDL and UACR. And insignificant association between mean TC, mean TG and mean HDL with UACR.

Key Words Type 2 Diabetes Mellitus, Lipid Profile, Diabetic Kidney Disease, Chronic Kidney Disease

INTRODUCTION

Diabetes Mellitus (DM) is a common metabolic disorder resulting from insufficient insulin secretion or insulin action that leads to elevated blood glucose levels [1]. The metabolic disorders associated with DM cause secondary pathophysiological changes in multiple organ systems that leading to acute and chronic complications [2]. Diabetic Kidney Disease (DKD) is one of the major chronic microvascular complications of diabetes and a leading cause of End-Stage Renal Disease (ESRD) and it is mostly due to Type 2 Diabetes Mellitus (T2DM) [3]. Hyperglycemia is the main driving force behind the development of chronic complications of diabetes, including DKD [4].

Insulin Resistance (IR) is frequently higher in patients with DM with microalbuminuria in the context of renal disease, which may accelerate the progression of Diabetic Nephropathy (DN) [5,6].

Lipid profile abnormalities play a main factor in the development of DKD other than cardiovascular disease (CVD) [7]. The lipoproteins in plasma are categorized into major groups by their size that is High-Density Lipoprotein (HDL), IDL, Low-Density Lipoprotein (LDL), VLDL, chylomicrons, chylomicron remnants and lipoprotein-a) [8]. Any abnormalities from reference ranges of lipid profile components are called dyslipidemia. The high risk of blood lipid and lipoprotein levels may occur because of many different reasons such as hereditary, obesity, age and lifestyle [9]. Periodic and early lipid profile measurements in diabetic patients are critical in avoiding the risks of CVD [10,11]. As a result of dyslipidemia, atherosclerosis which is a major risk factor for CVD is developing [12,13]. Severe atherosclerosis along with CVD is considered as a major factor leading to death in patients who suffer from Chronic Kidney Disease (CKD) [14]. Also, CKD is a major risk factor for CVD [15]. The higher mortality rates in severe CKD are highly associated with dyslipidemia among many other factors [16].

Aim

The study aims to examine the association between the lipid profile and DKD in T2DM patients and to evaluate the relationship between specific lipid parameters (such as TC, LDL, HDL and TG) and the severity of DKD in T2DM patients.

METHODS

Study Designs

A cross-sectional study was included 100 patients who had been diagnosed with T2DM attending Galiawa Teaching Center for diabetes and endocrinology. Data had been collected over six months, from June 2024 to January 2025. The sample size was calculated using Epi-Info software, with an expected frequency of 50%, a 95% confidence interval and a 5% acceptable margin of error. The study was approved by Ethical Committee of Faiha Specialized Diabetes, Endocrine and Metabolism Center (FDEMC) and conformed to the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Inclusion Criteria

All patients with type 2 DM.

Exclusion Criteria

We excluded Type 1 diabetes patients, patients with history of malignancy, chronic hepatitis and heart failure. Also, we excluded patients with acute diabetic complications, Diabetic patients on hemodialysis, pregnancy with T2DM, as well as other causes of CKD and urinary tract infections.

Data Collection

All patients were already diagnosed according to the American Diabetes Association (ADA) diagnostic criteria for diabetes and the type of diabetes was further identified by full history and clinical examination. The age, gender and details of diabetes treatment were retrieved from the patient's records and use of anti lipid medications (e.g., statins). Also history of hypertension, previous history of kidney problems and family history of diabetes or kidney problems and any symptoms related to kidney problems (e.g., swelling and changes in urination) were asked. Habitual factors were asked as dietary habits (e.g., frequency of consuming high-fat foods, fruits and vegetables), physical activity (e.g., sedentary, moderate, active) and smoking status (current, former, never). The duration of T2DM was calculated from the time of first diagnosis that was recalled by the patient and /or their relatives. The weight and height of each patient were measured wearing light clothes and shoes off, then the Body Mass Index (BMI) was calculated using the equation: weight (in kilogram)/height^2 (in meter^2). Blood Pressure (BP) was measured using a mercury sphygmomanometer from both patients' arms after sitting on a chair at least for 5 minutes and the highest measure was recorded depending on Korotkoff Phase I (for systolic BP) and Phase V (for diastolic BP).

Biochemical Tests

All recruited patients were fasting for at least 10 hours when peripheral venous blood samples were withdrawn. For each patient, withdrawn blood was transferred into a purple tube containing EDTA and a yellow tube containing a separation gel and coagulant which were labeled properly. Serum was separated immediately after centrifugation and laboratory biochemical tests were performed within 3-5 hours using electrochemiluminescence immunoassay kits. Roche Cobas c311 analyzer was used to measure the HbA1c, Fasting Plasma Glucose (FPG), fasting TG, total cholesterol, LDL and HDL cholesterol concentrations. The other tests included serum creatinine, spot urine for albumin-creatinine ratio (UACR), then estimated glomerular filtration rate (eGFR) calculated by CKD-EPI creatinine equation.

Data Analysis

The data were analyzed using appropriate statistical methods to determine the association between different variables. Descriptive statistics were used to summarize the sociodemographic and other characteristics of the participants. Statistical Package for the Social Sciences (SPSS) version 26 had been used for this purpose.

Definitions

Albuminuria is the presence of albumin (a type of protein) in the urine, is classified into three categories based on the severity of protein levels: A1 (normal to mildly increased) ACR less than 30mg/g. A2 (moderately increased) ACR between 30 to 300mg/g. A3 (severely increased) ACR greater than 300mg/g [16].

RESULTS

The general Characteristics of the Patients revealed that among the 100 participants, the median age was 57±9 years, ranging from 34 to 80 years. About 60% of the participants were females and 40 % were males. The mean HbA1c level during the time study was 8%, with values ranging from 5.5% to 13%, while the mean FBS was (175±64) mg/dl. The majority, 94 % of the participants have had a BMI of overweight and obese category, while only 6 % show normal values, the mean BMI was (31.1±4.3). The mean duration of diabetes among patients was (8.6±5.1) years. A high percentage (78.0%) of participants are on statin therapy while only (22.0%) of patients did not on statin therapy. About 74% of participants have other comorbidities and 26% of them did not have any comorbidities. Regarding lipid profiles, the mean triglyceride level was 219 mg/dL, while the mean total cholesterol level was 312 mg/dL. DKD was assessed using eGFR and microalbuminuria.

Table 1: General characteristics of 100 participants

Variables 1		Mean±Std
Age (years)		57±9
Male No. (%)		41 (41)
Female No. (%)		59 (59)
BMI		31.1±4.3
FBS (mg/dl)		175±64
A1c (%)		8.0±1.5
TC (mg/dl)		312±40
TG (mg/dl)		219±144
LDL (mg/dl)		116±35
HDL (mg/dl)		40±8.5
Serum creatinine (mg/dl)		1.15±0.64
Duration of Diabetes (years)		8.6±5.1
ACR	Proteinuria	No (%)
	A1	21 (21)
	A2	53 (53)
	A3	26 (26)
GFR	Stages	No (%)
	1	31 (31)
	2	37 (37)
	3a	18 (18)
	3b	10 (10)
	4	2 (2)
	5	2 (2)
On statin		78(78)
Not on statin		22(22)
With comorbidity		74 (74)
Without comorbidity		26 (26)

BMI: Body Mass Index, FBC: Fasting Blood Sugar, A1c: Hemoglobin A1c, TC: Total Cholesterol, TG: Triglyceride, LDL: Low Density Lipoprotein, HDL: High Density Lipoprotein, ACR: Albumin Creatinine Ratio, eGFR: Estimated Glomerular Filtration Rate, Stage 1: eGFR ≥90 ml/min, Stage 2: eGFR 60-90 ml/min, Stage 3a: eGFR 45-59 90 ml/min, Stage 3b: eGFR 30-44 ml/min, Stage 4: eGFR 15-29 ml/min, Stage 5: eGFR ≤15, A1: Microalbuminuria ≤30 mg/mg creatinine, A2: Microalbuminuria 30-300 mg/mg creatinine, A3: Microalbuminuria ≥300 mg/mg creatinine

Table 2: Represents the difference in the means of lipid profiles between ACR groups (total number of each group between 2 brackets)

Variables	A1 (21)	A2 (53)	A3 (26)	p-value
TC (Mean±Std)	185±`42	185±45	197±27	0.419
TG (Mean±Std)	192±89	240±139	199±57	0.136
LDL (Mean±Std)	97±40	113±32	136±26	0.000
HDL (mean±Std)	42±9	40±9	40±8	0,651

ACR: Albumin Creatinine Ratio, A1: Microalbuminuria ≤30 mg/mg creatinine, A2: Microalbuminuria 30-300 mg/mg creatinine, A3: Microalbuminuria ≥300 mg/mg creatinine, HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein, TC: Total Cholesterol, TG: triglyceride

Table 3: The comparison of the means of lipid profiles among the stages of eGFR groups (total number of each group between 2 brackets)

Variables	Stage 1 (31)	Stage 2 (37)	Stage 3a (18)	Stage 3b (10)	Stage 4 (2)	Stage 5 (2)	p-value
TC (Mean±Std)	180±45	194±41	190±21	187±57	200±7	180±0	0.826
TG (Mean±Std)	208±121	237±133	198±41	284±124	168±50	168±0	0.677
LDL (Mean±Std)	103±37	117±35	127±22	132±38	106±72	108±0	0.146
HDL (Mean±Std)	42±10	40±9	42±5	38±5	31±6	45±0	0.347

TC: Total Cholesterol, TG: Triglyceride, LDL: Low Density Lipoprotein, HDL: High Density Lipoprotein, eGFR: Estimated Glomerular Filtration Rate, Stage 1: eGFR ≥90 ml/min, Stage 2: eGFR 60-90 ml/min, Stage 3a: eGFR 45-59 90 ml/min, Stage 3b: eGFR 30-44 ml/min, Stage 4: eGFR 15-29 ml/min, Stage 5: eGFR ≤15

The most frequent category was stage A2, observed in 37 patients (37%). Additionally, 86% of participants had an eGFR within stages 1 to 3a. The mean serum creatinine levels was (1.15±0.64) mg/dl (Table 1).

The mean lipid profile values for TC, TG and HDL did not show statistically significant differences across the different ACR groups, with p-values of 0.419, 0.136 and 0.651, respectively. However, LDL levels showed a statistically significant difference between ACR groups A1, A2 and A3 (p-value = 0.000), with the highest mean observed in group A3 (136±26 mg/dL). Pairwise comparisons revealed a mean difference of 16 mg/dL between groups A1 and A2 (p-value = 0.15), 39 mg/dL between A1 and A3 (p-value = 0.000) and 22 mg/dL between A2 and A3 (p-value = 0.19). These results indicate that the significant difference is primarily driven by the elevated LDL levels in group A3 (Table 2).

To assess lipid levels across different stages of eGFR, the mean values of TC, LDL and HDL were calculated, showing no statistically significant differences. For further analysis, eGFR stages were grouped into two categories: ≥45 ml/min and <45 ml/min. Although a statistically significant p-value of 0.037 was observed, the difference in mean lipid levels was only 5 mg/dL, which is not considered clinically meaningful (Table 3 and 4).

Table 4: The difference in the means of lipid profiles between the 2 groups of eGFR (total number of each group between the 2 brackets)

Variables	eGFR ≥45 (86)	eGFR <45 (14)	p-value
TC (Mean±Std)	188±40	189±41	0.963
TG (Mean±Std)	210±102	237±173	0.534
LDL (Mean±Std)	116±36	112±25	0.673
HDL (Mean±Std)	42±10	40±8	0.037

TC: Total Cholesterol, TG: Triglyceride, LDL: Low Density Lipoprotein, HDL: High Density Lipoprotein, eGFR: Estimated Glomerular Filtration Rate

DISCUSSION

According to Kidney Disease Improving Global Outcomes (KDIGO) clinical practice guidelines for lipid management in CKD, adults with recently identified kidney disease should be evaluated for lipid profile abnormalities, as this condition, though not universal, is commonly present among people with DKD nephropathy [17].

Altered lipid profiles in T2DM are due to insulin resistance and defective insulin action on lipoprotein metabolism. Increased lipolysis will increase the synthesis of VLDL and triglyceride-rich LDL-C. It will also increase TG synthesis and promote the quick breakdown of HDL-C [17].

In our study, the most common pattern of lipid abnormality is a high level of serum TC. In a study done by Jayarama N, et al the most common pattern of dyslipidemia was combined dyslipidemia; High TG and low HDL were the most common [18].

The second most common dyslipidemia in our study was low HDL level, affecting 56% of patients. In a study done by Jayarama *et al.* [18] the second most common pattern of dyslipidemia was isolated low HDL level. These results were in comparison to our study.

In our study there was a significant correlation between mean LDL cholesterol in association with high levels of ACR. Yang *et al.* [19] was found in their study, similar to our study, that A3 microalbuminuria associated with high mean LDL level. Ravid *et al.* [20] found that mean LDL associated with high levels of ACR which is similar to our study. But there is no significant association between mean TC, mean HDL and mean TG with high levels of ACR in our study. Afghahi *et al.* [21] found that high ACR was associated with high mean TG level and mean HDL level. This is opposite to our study, maybe because most of the patients in our study are using anti lipid therapy.

A study done by Kolhar *et al.* [22] in India found a highly significant association of DKD with high mean TC but no significant association of ACR with HDL and it is also similar to our study. A study done by Jisieike-Onuigbo *et al.* [23] showed a significant association of ACR with high mean LDL.C and high mean TG. There is no significant association of ACR with TC and HDL-C. Which is somewhat agree with our study. A study done in Taiwan that strongly agree with our study showed that high ACR is significantly associated with high mean LDL but not with TC, HDL and TG levels [24].

The studies that differed from our study in their results may be due to the Dietary habits that differs from our country to other countries. As those on the vegetarian diet, TG and LDL were on the higher side, while those mostly consuming trans and saturated fat had total cholesterol on the higher side.

The association of lipid profile with various stages of CKD estimated by GFR in our study, there was a insignificant association between high mean TC, high mean LDL-C, low mean HDL-C and mean high TG with the severity of kidney disease as estimated by GFR. Zolezzi *et al.* [25] also reported increased TC, TG and decreased HDL in patients with various stages of CKD which is opposite to our study, because the sample in our study smaller than their study.

A previous paper from the Chronic Renal Insufficiency Cohort (CRIC) Study reported, which analyzed 3,939 adult patients with non-dialysis CKD of mean age 58.2 years and mean eGFR 44.9 mL/min/1.73 m2, that serum TG level is not independently associated with progression of CKD [26]. Which is similar to our study.

In a study done in india by Bhagwat *et al.* [27] they found HDL-C to be significantly low (20±11) mg/dL (p-value less than 0.001) in CKD stages and this is opposite to our study.

Lin *et al.* [28] study that done in united states showed that mean TG levels were negatively correlated with eGFR. Which is similar to our study.

The results of this study are different from the results of many other studies and this is may be due to most of the patients in our study were taking statins and fibrates for a long period and in a high doses.

CONCLUSIONS

Most if not all of the participants were elderly women suffering from being overweight and obese.

The high level of mean LDL and ACR points to a worrying clue of the deterioration of kidney function due to lipid abnormalities. There is a significant correlation between mean LDL and UACR. And insignificant association between mean TC, mean TG and mean HDL with UACR.

All stages of CKD were found to have low levels of HDL which necessitates proper treatment.

Limitations

This study has some limitations. Its cross-sectional design prevents establishing cause-and-effect relationships. Conducting the research at a single center may limit generalizability. the lack of follow-up limits understanding of disease progression and variations in laboratory data sources may introduce inconsistencies. Lastly, the impact of statin use was not fully analyzed.

Acknowledgement

I am deeply thankful to my wife Azheen Muhammed (Msc pharmacy) for her continuous support throughout this research work.

REFERENCES

[1] Asmamaw, Tadesse *et al.* "Early detection of renal impairment among patients with type 2 diabetes mellitus through evaluation of serum cystatin C in comparison with serum creatinine levels: a cross-sectional study." *Diabetes, Metabolic Syndrome and Obesity,* vol. 13, December 2020, pp. 4727-4735. https://www.tandfonline.com/doi/full/10.2147/DMSO.S279949.

[2] Zhou, Baoqin *et al.* "Clinical utility of serum cystatin c in predicting diabetic nephropathy among patients with diabetes mellitus: a meta-analysis." *Kidney and Blood Pressure Research,* vol. 41, no. 6, November 2016, pp. 919-928. https://karger.com/kbr/article/41/6/919/183569.

[3] Zhang, Xin-Xin *et al.* "Prevalence of diabetic nephropathy among patients with type 2 diabetes mellitus in China: A meta-analysis of observational studies." *Journal of Diabetes Research,* vol. 1, February 2020. https://onlinelibrary.wiley.com/doi/abs/10.1155/2020/2315607.

[4] Shahwan, Moyad Jamal *et al.* "Prevalence of diabetic nephropathy and associated risk factors among type 2 diabetes mellitus patients in Ramallah, Palestine." *Diabetes & Metabolic Syndrome: Clinical Research & Reviews,* vol. 13, no. 2, April 2019, pp. 1491-1496. https://www.sciencedirect.com/science/article/pii/S1871402119300505.

[5] Amorim, Rayne Gomes *et al.* "Kidney disease in diabetes mellitus: Cross-linking between hyperglycemia, redox imbalance and inflammation." *Arquivos Brasileiros de Cardiologia,* vol. 112, May 2019, pp. 577-587. https://www.scielo.br/j/abc/a/8DSdzvT5sd4tbTFM6N6PZCD/?format=html&lang=en.

[6] Pham, Hien *et al.* "Chronic kidney disease, insulin resistance, and incident diabetes in older adults." *Clinical Journal of the American Society of Nephrology,* vol. 7, no. 4, April 2012, pp. 588-594. https://journals.lww.com/CJASN/fulltext/2012/04000/Chronic_Kidney_Disease,_Insulin_Resistance,_and.11.aspx.

[7] Spoto, Belinda *et al.* "Insulin resistance in chronic kidney disease: a systematic review." *American Journal of Physiology-Renal Physiology,* vol. 311, no. 6, December 2016, pp. F1087-F1108. https://journals.physiology.org/doi/abs/10.1152/ajprenal.00340.2016.

[8] Thomas, Merlin C. *et al.* "Serum lipids and the progression of nephropathy in type 1 diabetes." *Diabetes Care,* vol. 29, no. 2, February 2006, pp. 317-322. https://diabetesjournals.org/care/article-abstract/29/2/317/26252.

[9] Feingold, K. R., and C. Grunfeld. *Introduction to lipids and lipoproteins. South Dartmouth: MDText. com* 2000.

[10] Magnussen, Costan G. *et al.* "Factors affecting the stability of blood lipid and lipoprotein levels from youth to adulthood: evidence from the Childhood Determinants of Adult Health Study." *Archives of Pediatrics & Adolescent Medicine,* vol. 165, no. 1, 2011, pp. 68-76. https://jamanetwork.com/journals/jamapediatrics/article-abstract/384173.

[11] Freitas, Roberto Wagner Júnior Freire de *et al.* "Análisis del perfil lipídico en una población de estudiantes universitarios." *Revista Latino-Americana de Enfermagem,* vol. 21, October 2013, pp. 1151-1158. https://www.scielo.br/j/rlae/a/bm7pVVb8PSq6Xws7kRTsrdj/?lang=es.

[12] Brandão, Maria Piedade *et al.* "Impacto de la exposición académica en el estado de salud de estudiantes universitarios." *Revista de Saúde Pública,* vol. 45, 2010, pp. 49-58. https://www.scielosp.org/article/ssm/content/raw/?resource_ssm_path=/media/assets/rsp/v45n1/2035.pdf.

[13] Scheffer, Peter G. *et al.* "Increased plasma apolipoprotein C-III concentration independently predicts cardiovascular mortality: The Hoorn Study." *Clinical Chemistry,* vol. 54, no. 8, August 2008, pp. 1325-1330. https://academic.oup.com/clinchem/article-abstract/54/8/1325/5628538.

[14] Bobik, Alex. "Apolipoprotein CIII and atherosclerosis: beyond effects on lipid metabolism." *Circulation,* vol. 118, no. 7, August 2008, pp. 702-704. https://www.ahajournals.org/doi/full/10.1161/CIRCULATIONAHA.108.794081.

[15] Go, A.S., G.M. Chertow, D. Fan, C.E. Mcculloch and C.Y. Hsu, "Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization.." *Engl. J. Med.,* vol. 351, 2004, pp. 1296-1305. http://dx.doi.org/10.1056/nejmoa041031.

[16] Schiffrin, Ernesto L. *et al.* "Chronic kidney disease: effects on the cardiovascular system." *Circulation,* vol. 116, no. 1, July 2007, pp. 85-97. https://www.ahajournals.org/doi/full/10.1161/CIRCULATIONAHA.106.678342.

[17] Trovati, Mariella, and Franco Cavalot. "Optimization of hypolipidemic and antiplatelet treatment in the diabetic patient with renal disease." *Journal of the American Society of Nephrology,* vol. 15, no. 1, January 2004, pp. S12-S20. https://journals.lww.com/JASN/fulltext/2004/01001/Optimization_of_Hypolipidemic_and_Antiplatelet.3.aspx.

[18] Jayarama, N. *et al.* "Prevalence and pattern of dyslipidemia in type 2 diabetes mellitus patients in a rural tertiary care centre, southern India." *Global Journal of Medicine & Public Health,* vol. 1, no. 3, 2012, pp. 24-28.

[19] Yang, Xilin *et al.* "Effects of albuminuria and renal dysfunction on development of dyslipidaemia in type 2 diabetes-the Hong Kong Diabetes Registry." *Nephrology Dialysis Transplantation,* vol. 23, no. 9, 2008, pp. 2834-2840.

[20] Ravid, Mordchai *et al.* "Main risk factors for nephropathy in type 2 diabetes mellitus are plasma cholesterol levels, mean blood pressure, and hyperglycemia." *Archives of Internal Medicine,* vol. 158, no. 9, May 1998, pp. 998-1004. https://jamanetwork.com/journals/jamainternalmedicine/article-abstract/205097.

[21] Afghahi, Henri *et al.* "Risk factors for the development of albuminuria and renal impairment in type 2 diabetes-the Swedish National Diabetes Register (NDR)." *Nephrology Dialysis Transplantation,* vol. 26, no. 4, September 2010, pp. 1236-1243. https://academic.oup.com/ndt/article-abstract/26/4/1236/1878919.

[22] Kolhar U. *et al.* "International Journal of Advances in Medicine." *International Journal of Advances in Medicine,* vol. 4, no. 6, 2017, pp. 1513-1516.

[23] Jisieike-Onuigbo, N. N. *et al.* "Prevalence of dyslipidemia among adult diabetic patients with overt diabetic nephropathy in Anambra state South-East Nigeria." *Nigerian Journal of Clinical Practice,* vol. 14, no. 2, 2011, pp. 171-175. https://www.ajol.info/index.php/njcp/article/view/74512.

[24] Huang, Po-Hsun *et al.* "2022 Taiwan lipid guidelines for primary prevention." *Journal of the Formosan Medical Association,* vol. 121, no. 12, 2022, pp. 2393-2407. https://www.sciencedirect.com/science/article/pii/S0929664622002157.

[25] Zolezzi M. "Management of dyslipidemia in renal disease and transplantation." *Saudi Journal of Kidney Diseases and Transplantation,* vol. 17, 2006, pp. 129-136.

[26] Rahman, Mahboob *et al.* "Relation of serum lipids and lipoproteins with progression of CKD: The CRIC study." *Clinical journal of the American Society of Nephrology,* vol. 9, no. 7, 2014, pp. 1190-1198. https://journals.lww.com/cjasn/fulltext/2014/07000/Relation_of_Serum_Lipids_and_Lipoproteins_with.9.aspx.

[27] Bhagawat R. *et al.* "Lipid abnormality in chronic renalfailure." *Indian Journal of Clinical Biochemistry,* vol. 12, no. 1, January 1997, pp. 81-85.

[28] Lin, Jennie *et al.* "Plasma lipoprotein (a) levels are associated with mild renal impairment in type 2 diabetics independent of albuminuria." *PLoS One,* vol. 9, no. 12, December 2014. https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0114397.