

Original Article

A Retrospective Cross-Sectional Study To Assess The Relationship Between Renal Dysfunction And Lipid Profile Secondary To Cardiovascular Disease

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Abstract

Objective: To assess the relationship between renal dysfunction and lipid profiles in patients with cardiovascular disease.

Methodology: This retrospective cross-sectional study was conducted at the Wazirabad Institute of Cardiology, Gujranwala, Pakistan. Patients with cardiovascular disease who visited the Wazirabad Institute of Cardiology between 1st April and 30 June 2022 were included in the study. Data were entered and analysed using SPSS 26. Spearman's and Pearson's correlation coefficients were used to determine the correlations. All results were calculated at a 95% confidence interval.

Results: A total of 409 patients with cardiovascular disease were included. Of these, 254 were men, and 155 were women. The mean age of the patients was 53.71 ± 12.17 years. LDL was only significantly correlated with blood urea but not with creatinine. HDL, cholesterol, and triglyceride levels were not significantly correlated with urea and creatinine levels.

Conclusion: Blood urea, a marker of renal dysfunction, and low-density lipoprotein, a marker of lipid profile, were strongly correlated in this study. Therefore, there was a relationship between renal dysfunction and the lipid profile. The strongest significant statistical correlation was between urea and LDL. However, in the female population, LDL-C, creatinine, triglycerides, and cholesterol were also found to be significant. All other variables, such as HDL, Cholesterol, and triglyceride levels, were not correlated with blood urea levels. In addition, none of the variables, including HDL, LDL, Cholesterol, Triglycerides, were correlated with creatinine, a variable of renal dysfunction.

Keywords: Cardiovascular Disease, Renal Dysfunction, Lipid Profile

Introduction

Because of their high morbidity and significant mortality rates among working-age individuals, cardiovascular diseases (CVD) are considered socially relevant conditions and are ranked number 1 due to high morbidity and mortality rates worldwide.^{1,2} The primary cause of death linked to chronic kidney disease (CKD) is also cardiovascular disease (CVD).³

As the main cause of renal dysfunction in heart failure, the pathophysiological aspects have shown that heart failure may result in decreased cardiac output and renal perfusion.⁴ CKD often leads to end-stage renal disease (ESRD), which is strongly correlated with increased cardiovascular morbidity and mortality rates.⁵ The death rate is higher in patients with CKD and CVD than in those with ESRD.⁶ Lipid and lipoprotein particles are essential for proper heart and vascular function.⁷

Dyslipidaemia has emerged as a key complicating factor for renal impairment in heart failure.⁸ Dyslipidaemia, a significant pathogenic factor of atherosclerosis and a modifiable risk factor for cardiovascular and cerebrovascular diseases, is frequently observed in patients with chronic kidney disease (CKD).⁹ Both CVD and chronic kidney disease (CKD) contribute to the development of inflammation, which alters lipid balance.¹⁰

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Data Availability Statement: Upon reasonable request from the corresponding author, the authors will provide raw data supporting the results of this article without undue hesitation. Due to ethical limitations regarding patient privacy and the safeguarding of sensitive clinical data, the data are not accessible to the general public.

Institutional Review Board

Approval

REG/GBI/22/AHS-63

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There is evidence that atherosclerotic cardiovascular diseases and subsequent cardiovascular events are caused by low blood-density lipoprotein cholesterol (LDL-C) levels and other lipid risk factors, including triglycerides, low HDL cholesterol (HDL-C), and non-HDL-C.^{11,12} Creatinine and serum urea (also known as blood urea nitrogen) have been used as twin indicators of renal filtration. However, a recent study has reinterpreted urea as an active metabolic reactant rather than a passive waste product. Whereas creatinine is a constant consequence of muscle hypertrophy, urea concentrations vary in response to hepatic protein metabolism and systemic oxidative stress, both of which are crucial for the development of cardiovascular disease (CVD).¹³

The increased risk of CVD was caused by lower levels of high-density lipoprotein cholesterol (HDL-C) and higher levels of conventional lipid indices, such as total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG).^{14,15} Hypertriglyceridaemia and low levels of high-density lipoprotein cholesterol (HDL-C), two lipid abnormalities that are hallmarks of chronic kidney disease (CKD), are significant risk factors for cardiovascular diseases.¹⁶ The patients have elevated plasma triglyceride levels, decreased HDL-C levels, and problems with cholesterol transport. The decreased activity of the reverse cholesterol pathway and decreased HDL-C production are additional variables that may lead to atherosclerotic coronary artery disease in patients with ESRD.¹⁷

This study aimed to assess the relationship between renal dysfunction and lipid profiles in patients with cardiovascular disease.

Methodology

This retrospective cross-sectional study was conducted at the Wazirabad Institute of Cardiology, Gujranwala, Pakistan. Patients with cardiovascular disease who visited the Wazirabad Institute of Cardiology, Punjab, Pakistan, were included in the study. Retrospective data were collected between 1st April and 30 June 2022 from the hospital records. Demographic characteristics and lab test reports, including lipid profiles and renal function tests, were gathered from 409 patients with cardiovascular disease, including males and females. This study was conducted and reported according to the STROBE guidelines for observational studies.

Records of patients with cardiovascular disease, including males and females of any age, were selected. Data collection was performed after approval (reference no. REG/GRT/22/AHS-63). Biochemical reports of 470 patients with cardiovascular disease were obtained. Data from both sexes and all ages were included. A total of 409 patients with cardiovascular disease were included in the study. Records of 61 patients with missing information were excluded from the study. Patients were excluded if they had a missing history of medications and test reports. Data were collected from the Wazirabad Institute of Cardiology, Gujranwala. Demographic data were used. The biochemical reports included Urea, Creatinine, HDL, LDL, Triglycerides, and Cholesterol. The data were collected from the previous reports and the hospital management system.

To provide a representative clinical cohort, selection bias was reduced by using a successive sampling technique for all patients who met the predetermined inclusion criteria during the study period. Strict exclusion criteria and multivariate statistical adjustments were used to address confounding variables, such as age and pre-existing comorbidities. Together, these actions guarantee the dependability of the observed connections and improve the internal validity of our conclusions.

Sample size estimation was done through Fisher's transformation method.¹⁸

$$n = 3 + \frac{(Z_{1-\alpha/2} + Z_{1-\beta})^2}{(0.5 \times \ln(\frac{1+r}{1-r}))^2}$$

$r=0.243$,¹⁹ $Z_{(1-\alpha/2)}=1.96$ at 95% confidence interval and 80% study power were used for sample size estimation, and at least 215 samples were appropriate; however, for the reliability and to improve the accuracy of the data, 409 records of patients were selected. The study depends strictly on standard biochemical archives and does not use registered antibodies, cell lines, or organism models. Research Resource Identifiers (RRIDs) are absent.

This study was conducted after approval from the Institutional Review Board (IRB) of the University of Lahore, Gujrat Campus, under reference no. REG/GRT/22/AHS-63 and IRB number-63, dated 28-03-2022.

An electronic health record system was used to extract all patient data retrospectively. All personally identifiable information (PII) was eliminated before analysis to preserve participant anonymity. Each record was assigned a unique, non-traceable, study-specific alphanumeric code to facilitate statistical processing while preventing any potential re-identification of individual participants. In compliance with regional data protection laws, this procedure was examined and authorised, waiving the requirement for informed consent. The researchers have revised the manuscript to clearly state that the University of Lahore's Institutional Review Board formally waived the need for informed patient consent because the study was retrospective and used anonymised data in compliance with ethical transparency and open science guidelines.

The quantitative variables were age, sex, urea, creatinine, high-density lipoprotein (HDL), low-density lipoprotein (LDL), Cholesterol, Triglycerides, and other variables.

Data were analysed using the IBM Statistical Packages for Social Sciences (SPSS), Version 26. For the descriptive analysis, frequencies and percentages were calculated for qualitative variables, and means and standard deviations were calculated for quantitative data. The Shapiro-Wilk test was used to assess the normality of the data. When the data of both groups were assessed separately, the data were non-normal, and Spearman's correlation was applied; however, when the data were assessed collectively, they appeared normal, and the Pearson Correlation Coefficient was applied to assess the relationship between renal dysfunction and lipid profile variables. A p-value ≤ 0.05 was considered statistically significant.

Results

A relationship between renal dysfunction and lipid profiles has been established. A total of 409 patients with cardiovascular disease were enrolled in this study. Of these, 254 were men, and 155 were women. The biochemical reports consisted of urea, creatinine, high-density lipoprotein (HDL), low-density lipoprotein (LDL), Cholesterol, Triglycerides, and other variables.

Table 1 shows the age and sex characteristics and the mean and standard deviation of the urea, creatinine, HDL, LDL, cholesterol, and triglyceride levels.

Table 1: Demographic characteristics and lipid profile of patients (n=409)

Characteristics	Unit/Responses	n(%)	Mean $\pm$ S.D
Age	Years		53.71 $\pm$ 12.17
Gender	Male	254(62.10)	
	Female	155(37.90)	
Urea	mg/dl		35.47 $\pm$ 19.74
Creatinine	mg/dl		0.97 $\pm$ 0.47
HDL-C	mg/dl		40.04 $\pm$ 10.23
LDL-C	mg/dl		118.4 $\pm$ 28.43
Cholesterol	mg/dl		176.16 $\pm$ 35.26
Triglycerides	mg/dl		144.02 $\pm$ 64.53

No relationship was found between any kind of HDL (the variable of lipid profile) and urea and creatinine (the variables of renal dysfunction) in this study. No relationship between cholesterol (a variable of lipid profile) and urea and creatinine (variables of renal dysfunction) was found. No relationship between triglycerides (a variable of lipid profile) and urea and creatinine (variables of renal dysfunction) was found in this study. When the relationship between LDL (a variable of lipid profile) and urea and creatinine (variables of renal dysfunction) was examined, only the association of LDL with urea was significant. LDL is known as bad cholesterol and is the main risk factor for cardiovascular disease. Table 2 shows the association of urea and creatinine (the variables of renal dysfunction) with HDL, LDL, Cholesterol, Triglycerides (the lipid profile variables), including their values and p-values.

Table 2: Association between renal dysfunction and lipid profile variables.

Association	Male(n=254)	Female (n= 155)	Total (n=409)
	r(p-value) Spearman's		r(p-value) Pearson
HDL-C(mg/dl) and Urea(mg/dl)	0.106(0.093)	0.014(0.860)	0.00(0.988)
HDL-C(mg/dl) and Creatinine(mg/dl)	-0.088(0.163)	-0.057(0.482)	-0.08(0.109)
LDL-C(mg/dl) and Urea(mg/dl)	0.255(<0.001*)	0.098(0.258)	0.11(0.027*)
LDL-C(mg/dl) and Creatinine(mg/dl)	-0.022(0.722)	0.162(0.044*)	0.04(0.436)
Cholesterol(mg/dl) and Urea(mg/dl)	0.121(0.055)	0.117(0.149)	0.07(0.148)
Cholesterol(mg/dl) and Creatinine(mg/dl)	-0.068(0.283)	0.029(0.724)	-0.04(0.464)
Triglycerides(mg/dl) and Urea(mg/dl)	0.015(0.817)	0.087(0.284)	-0.02(0.654)
Triglycerides(mg/dl) and Creatinine(mg/dl)	0.067(0.287)	0.173(0.031*)	0.05(0.327)

(*) Indicates statistical significance at $p \leq 0.05$.

Discussion

The study finds the relationship between renal dysfunction and lipid profile variables. The high-density lipoprotein (the variable of lipid profile) was not significantly correlated with urea and creatinine (the variables of renal dysfunction), with p value = 0.988, p value = 0.109, respectively, in this conducted research; no relation was found between them. The LDL (the variable of lipid profile) was significantly correlated with urea (the variable of renal dysfunction) with p -value = 0.027, and the LDL is not significantly correlated with creatinine (the variable of renal dysfunction) with p -value = 0.436 in the overall population, but both variables are significantly correlated in the female population only, with a p -value of 0.044. The Cholesterol (the variable of lipid profile) was also not significantly correlated with urea and creatinine (the variables of renal dysfunction), with p -value = 0.148, p -value = 0.464, respectively; no relation was found between them. The Triglycerides (the variable of lipid profile) were also not significantly correlated with urea and creatinine (the variables of renal dysfunction), with p value = 0.654, p value = 0.327, respectively; hence, no relation was found between them.

There was a strong relationship between renal dysfunction and lipid profile, as both are risk factors for cardiovascular disease. This study included 409 patients with cardiovascular disease, and the relationship between renal dysfunction and lipid profiles was studied. This study examined the association between different factors linked to renal dysfunction and lipid profiles, such as Urea, Creatinine, HDL-C, LDL-C, Cholesterol, and Triglycerides. This study included a large sample size compared to other studies.

In our study, the mean values of urea, creatinine, HDL-C, LDL-C, cholesterol, and triglycerides were 35.47 ± 19.74 , 0.97 ± 0.47 , 40.04 ± 10.23 , 118.4 ± 28.43 , 176.16 ± 35.26 , and 144.02 ± 64.53 , respectively. In another study, the values of urea, creatinine 1.1, HDL-C 40, LDL-C, cholesterol, and triglycerides were 43, 1.1, 40, 137.26 ± 43.5 , 192.6 ± 48.27 , and 125 mg/dl, respectively. These values are in mg/dl, and the values are slightly different from those in our study, but all the values are close to our results. In our study, we determined the significance in males and females separately and then determined the overall association between renal dysfunction and lipid profile secondary to CVD. In our study, HDL-C, LDL-C, cholesterol, and triglyceride levels were directly or indirectly associated with serum creatinine levels in men, but the associations were not significant. In females, LDL-C and triglyceride levels were significant. Overall, these factors are associated with renal dysfunction and lipid profiles secondary to CVD, but are not significant. In another study, which defined the overall association between these factors, TG-CR, CH-CR, and HDL-CR were found to be significant.²⁰

In another study, the mean values of HDL-C, LDL-C, cholesterol, and triglycerides were 31.82 ± 9.735 , 71.42 ± 21.987 , 131.45 ± 33.878 , and 120.65 ± 72.097 mg/dl, respectively. The mean values for these four parameters were lower than those in our study.²¹

In another study, the mean values of HDL-C, LDL-C, cholesterol, triglycerides, and creatinine were 36.0 ± 25.1 , 103 ± 30.8 , 170 ± 36.8 , 155 ± 70.2 , and 9.04 ± 3.10 mg/dl, respectively. In comparison, most of the mean values were very close to those in our study.²² Recent studies have shown that urea is a precursor to carbamylated LDL (cLDL), which directly causes plaque development rather than being a passive waste product.²³ A study demonstrated that while creatinine frequently indicates late-stage problems, blood urea nitrogen (BUN) is an independent predictor of new-onset cardiovascular disease.²⁴ In our study, we found an association between Urea and HDL, LDL, Triglycerides, and Cholesterol. In males and overall, only urea-LDL was found to be significant. Compared to another study, urea-cholesterol and urea-LDL were significant. Urea-LDL was found to be significant in both studies. However, there was a difference in the sample size between the two studies.²⁵ In our study, LDL-C, creatinine, and triglyceride levels were found to be significant only in female patients. According to another study, these were found to be significant in the female population; this is because men's creatinine levels are significantly diluted by differences in muscle mass, as creatinine is released from the muscles. The association between creatinine levels and lipids such as LDL-C is significantly closer and statistically significant in women, who generally have lower and more stable muscle mass. This is because creatinine levels are a more strict reflection of metabolic and renal health.²⁶

Limitations:

A large amount of data was used in this study. The data used in this study applied to the study objectives. Previous studies have not linked the association between renal dysfunction and lipid profile in patients with CVD, but this study shows the association between renal dysfunction and lipid profile. The study was retrospective, and therefore, past data were used. Some data, such as BMI, were available; however, anthropometric measurements such as weight and height were not included in the patients' reports. This study also could not obtain the dietary history of the patient.

Conclusions

Blood urea, a marker of renal dysfunction, and low-density lipoprotein, a marker of the lipid profile, were strongly correlated. Therefore, there was a relationship between renal dysfunction and lipid profiles. The strongest significant statistical correlation was between urea and LDL. However, in the female population, LDL-C, creatinine, triglycerides, and cholesterol were also found to be significant. All other variables, such as HDL, Cholesterol, and triglyceride levels, were not correlated with blood urea levels. In addition, none of the variables, including HDL, LDL, Cholesterol, Triglycerides, were correlated with creatinine, a variable of renal dysfunction.

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Contributions:

AR SK - Conception, Design

AR QU - Acquisition, Analysis, Interpretation

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All authors approved the final version to be published & agreed to be accountable for all aspects of the work.

References

1. Becheva MSV, Kirkova-Bogdanova AG, Ivanova SA, Atanasov PJ, Chaneva MS, Petkova VB. Prevention of cardiovascular diseases. *Pharmacia*. 2023;70. <https://doi.org/10.3897/pharmacia.70.E114071>.
2. Ciumărnean L, Milaciu MV, Negrean V, Orășan OH, Vesa SC, Sălăgean O, et al. Cardiovascular risk factors and physical activity for the prevention of cardiovascular diseases in the elderly. *Int J Environ Res Public Health*. 2022;19(1):207. <https://doi.org/10.3390/ijerph19010207>
3. Lecamwasam A, Mansell T, Ekinci EI, Saffery R, Dwyer KM. Blood Plasma Metabolites in Diabetes-Associated Chronic Kidney Disease: A Focus on Lipid Profiles and Cardiovascular Risk. *Front Nutrition*. 2022;9. <https://doi.org/10.3389/fnut.2022.821209>.
4. Greene SJ, Butler J, Albert NM, DeVore AD, Sharma PP, Duffy CI, et al. Medical Therapy for Heart Failure With Reduced Ejection Fraction: The CHAMP-HF Registry. *J Am Coll Cardiol*. 2018;72. <https://doi.org/10.1016/j.jacc.2018.04.070>.
5. Batra G, Ghukasyan Lakic T, Lindbäck J, Held C, White HD, Stewart RAH, et al. Interleukin 6 and Cardiovascular Outcomes in Patients with Chronic Kidney Disease and Chronic Coronary Syndrome. *JAMA Cardiol*. 2021;6. <https://doi.org/10.1001/jamacardio.2021.3079>.
6. Subbiah AK, Chhabra YK, Mahajan S. Cardiovascular disease in patients with chronic kidney disease: A neglected subgroup. *Heart Asia*. 2016;8. <https://doi.org/10.1136/heartasia-2016-010809>.
7. Soppert J, Lehrke M, Marx N, Jankowski J, Noels H. Lipoproteins and lipids in cardiovascular disease: from mechanistic insights to therapeutic targeting. *Adv Drug Deliv Rev*. 2020;159:4–33. <https://doi.org/10.1016/j.addr.2020.07.019>
8. Gersei I, Krämer BK, Scharnagl H, Stojakovic T, März W. Renal function, N-terminal Pro-B-Type natriuretic peptide, propeptide big-endothelin and patients with heart failure and preserved ejection fraction. *Peptides (NY)*. 2019;111. <https://doi.org/10.1016/j.peptides.2018.04.003>.
9. Zhang A, Deng W, Zhang B, Ren M, Tian L, Ge J, et al. Association of lipid profiles with severity and outcome of acute ischemic stroke in patients with and without chronic kidney disease. *Neurological Sciences*. 2021;42. <https://doi.org/10.1007/s10072-020-04791-x>.
10. Reiss AB, Voloshyna I, De Leon J, Miyawaki N, Mattana J. Cholesterol metabolism in chronic kidney disease. *Am J Kidney Dis*. 2015;66(6):1071–1082. <https://doi.org/10.1053/j.ajkd.2015.06.028>

11. Ho LT, Yin WH, Chuang SY, Tseng WK, Wu YW, Hsieh IC, et al. Determinants for achieving the LDL-C target of lipid control for secondary prevention of cardiovascular events in Taiwan. *PLoS One*. 2015;10. [https://doi.org/ 10.1371/journal.pone.0116513](https://doi.org/10.1371/journal.pone.0116513).
12. Yin WH, Wu CC, Chen JW. Registry of lipid control and the use of lipid-lowering drugs for secondary prevention of cardiovascular events in patients with established atherosclerotic disease in Taiwan: Rationality and methods. *Int J Gerontol*. 2012;6. [https://doi.org/ 10.1016/j.ijge.2012.09.001](https://doi.org/10.1016/j.ijge.2012.09.001).
13. Laville SM, Couturier A, Lambert O, Metzger M, Mansencal N, Jacquelinet C, et al. Urea levels and cardiovascular disease in patients with chronic kidney disease. *Nephrology Dialysis Transplantation*. 2022;38:184. [https://doi.org/ 10.1093/ndt/gfac045](https://doi.org/10.1093/ndt/gfac045). Cited in PMID: 35544273.
14. Gu X, Li Y, Chen S, Yang X, Liu F, Li Y, et al. Association of Lipids With Ischemic and Hemorrhagic Stroke. *Stroke*. 2019;50. [https://doi.org/ 10.1161/strokeaha.119.026402](https://doi.org/10.1161/strokeaha.119.026402).
15. Sun L, Clarke R, Bennett D, Guo Y, Walters RG, Hill M, et al. Causal associations of blood lipids with risk of ischemic stroke and intracerebral hemorrhage in Chinese adults. *Nat Med*. 2019;25. [https://doi.org/ 10.1038/s41591-019-0366-x](https://doi.org/10.1038/s41591-019-0366-x).
16. Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: global dimension and perspectives. *Lancet*. 2013;382(9888):260–272. [https://doi.org/10.1016/S0140-6736\(13\)60687-X](https://doi.org/10.1016/S0140-6736(13)60687-X)
17. Patil S, Kumar VA, Subramani S. To evaluate Lipid Profiles in Patients with Chronic Kidney Disease in RajaRajeswari Medical College and Hospital, Bengaluru, Karnataka, India. *The Journal of Medical Sciences*. 2018;4. [https://doi.org/ 10.5005/jp-journals-10045-0080](https://doi.org/10.5005/jp-journals-10045-0080).
18. Cochran WG. Sampling techniques. 3rd ed. New York (NY): John Wiley & Sons; 1977.
19. Zhang H, Shi S, Zhao XJ, Wang JK, Liu ZW, Liu FQ, et al. Association Between the Lipid Profile and Renal Dysfunction in the Heart Failure Patients. *Kidney Blood Press Res*. 2019;44:52–61. [https://doi.org/ 10.1159/000498834](https://doi.org/10.1159/000498834). Cited in PMID: 30808836.
20. Purwowiyoto SL, Lasanudin HF, Setianto B, Surya SP, Diwirya WC. Correlation Between Lipid Profile and Kidney Function in Ambulatory Heart Failure Patient. *Mutiara Medika: Jurnal Kedokteran dan Kesehatan*. 2021;22. [https://doi.org/ 10.18196/mmjkk.v1i1.9969](https://doi.org/10.18196/mmjkk.v1i1.9969).
21. Shanmugam L, Kadavanu TM, Green SR, Dutta TK, Radhakrishnan H, Ramachandrappa AK, et al. A comparative study of lipid profile and cardiovascular risk biomarkers among chronic haemodialysis patients and healthy individuals. *Journal of Clinical and Diagnostic Research*. 2016;10. [https://doi.org/ 10.7860/JCDR/2016/21897.8523](https://doi.org/10.7860/JCDR/2016/21897.8523).
22. Mohammed MA, Mohammed IA, Ismail AM, Ali AM, Dafaalla MH, Mohammed RK, et al. Association of Serum Lipid Profile Levels in Chronic Kidney Disease Patients with Cardiovascular Disease a case control study, Khartoum State. *Napata Sci J*. 2022;1. [https://doi.org/ 10.53796/nsj123](https://doi.org/10.53796/nsj123).
23. Saar-Kovrov V, Pawlowska A, Guillot A, Gijbels MJJ, Sluimer JC, Temmerman L, et al. Protein carbamylation in atherosclerotic plaques correlates with uremia and disease progression, localizing predominantly to foam cells. *Front Immunol*. 2025;16:1532250. [https://doi.org/ 10.3389/fimmu.2025.1532250](https://doi.org/10.3389/fimmu.2025.1532250). Cited in PMID: 40503238.
24. Lai W, Chen X, Wang L, Wu L, Li X, Zhou B. Association between LDL/HDL ratio and hypertension in Chinese middle-aged and older adults: a cross-sectional and longitudinal analysis based on CHARLS LDL/HDL ratio and hypertension. *Front Endocrinol (Lausanne)*. 2025;16:1484318. [https://doi.org/ 10.3389/fendo.2025.1484318](https://doi.org/10.3389/fendo.2025.1484318).
25. Baqi HR, K. Rostam SR. The Correlation Between Lipid Profile and Renal Function Tests in Patients with Cardiovascular Disease in Erbil city, Kurdistan Region of Iraq. *Kurdistan Journal of Applied Research*. 2020; [https://doi.org/ 10.24017/science.2020.2.5](https://doi.org/10.24017/science.2020.2.5).
26. De La Mata NL, Hedley JA, Webster AC, Sullivan MK, Rosales BM, Mark PB, et al. Sex differences in cardiac risk and kidney function: serum creatinine versus cystatin C. *BMC Medicine*. 2025 Dec 30. [https://doi.org/ 10.1186/s12916-025-04588-9](https://doi.org/10.1186/s12916-025-04588-9)