Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 16 May 2026
Revised 26 June 2026
Accepted 10 July 2026

Natural Resources for Human Health 2026, Vol. 6(7s): 1011–1019

KEYWORDS:
renal-related biochemical markers; insulin resistance; HOMA-IR; atherogenic dyslipidemia; hsCRP; case-control study

Metabolic, Inflammatory, and Atherogenic Profiles Associated with Renal-Related Biochemical Markers: An Integrated Case-Control Analysis

Saif Salah Abdul Hassan[1*]

[1]Department of Computer, College of Basic Education, Al-Mustansiriyah University, Baghdad, Iraq.
*Correspondence: Email: saif.s.abd@uomustansiriyah.edu.iq,
ORCID: https://orcid.org/0009-0005-8090-0774

ABSTRACT: Metabolic, inflammatory, and atherogenic abnormalities may coexist with changes in renal-related biochemical markers. This retrospective case-control analysis evaluated an integrated biochemical profile in 379 archived records, comprising 173 cases and 206 controls. Because data availability varied across variables, analyses used available observations rather than a single common denominator. Sodium, potassium, chloride, magnesium, insulin, HOMA-IR, hsCRP, and conventional and derived lipid indices were evaluated. The archived case-side creatinine field consisted of zero-coded values that could not be verified as physiologic measurements; therefore, creatinine was not used for clinical interpretation or multivariable modelling in the corrected analysis. Potassium and HOMA-IR showed significant between-group differences, while several lipid and atherogenic indices provided the broadest separation. hsCRP did not differ significantly between groups and showed no significant association with the evaluated renal-related electrolyte markers. Overall, the dataset supports a multidimensional metabolic and atherogenic pattern associated with selected renal-related biochemical markers, while renal-specific interpretation remains limited by the quality and completeness of the archived renal data.

1. INTRODUCTION

Chronic kidney disease (CKD) and renal-risk states remain a large clinical and public health problem as biochemical changes may take many years to progress to clinical disease [1]. Current recommendations for kidney counseling therefore indicate a multi-dimensional risk assessment, not only relying on a single laboratory parameter, and kidney vulnerability might exist before there is much clinical kidney dysfunction, especially in populations with metabolic disturbance [1]. In this context, an integrated biochemical approach is significant in particular with regard to the identification of early renal-risk patterns.

Renal injury is associated with various pathways, one of which is insulin resistance, which has become a biologically plausible and clinically important pathway. Experimental and clinical data suggest that insulin resistance is associated with endothelial dysfunction, oxidative stress, abnormal tubular sodium handling, microvascular instability and maladaptive hemodynamic responses, which have the potential to promote renal injury [6]. Epidemiologic and cohort data more recently also indicate that insulin-resistance indices and associated surrogates are linked to CKD prevalence and incident CKD as well as diabetic kidney disease phenotypes [5,8,9]. Therefore, insulin resistance itself is not only a metabolic disorder, but may also be a factor for renal-risk stratification.

Another significant pathway between metabolic dysfunction and the vulnerability of the kidneys is dyslipidemia. There is increasing evidence that triglyceride-rich lipoproteins, decreased HDL related protection, non-HDL atherogenic burden and composite ratios (e.g., TG/HDL-C) are linked to the decline in renal function, progression of CKD and DN [4,7,10-12]. These

relationships are biologically plausible as the atherogenic lipids can lead to glomerular and tubulointerstitial injury via lipotoxicity, endothelial dysfunction, oxidative stress, inflammatory signaling, and HDL dysfunction [7,10,12]. Thus, lipid-derived indices might offer clinically valuable information in addition to discrete traditional lipid measures.

Inflammation also plays a role in the context of renal-risk, specifically low-grade systemic inflammation as indicated by high sensitivity C-reactive protein (hsCRP). There have been indications that chronic hsCRP elevation could predict further loss of renal function and proteinuria, particularly among metabolically-compromised individuals [3]. Concurrently, mechanistic and translation studies suggest that CRP could be involved in renal inflammation and fibrosis processes, but associations are not consistent across populations and study designs [2]. This indicates that inflammation could be a modifier of renal risk and not necessarily a dominant determinant in all datasets.

Given this background, combining renal-related biochemical markers with metabolic, inflammatory, and atherogenic variables may provide a broader description of biochemical vulnerability than considering each domain in isolation. The present study therefore aimed to characterize the case-control pattern across these domains and to examine associations between renal-related electrolyte markers and metabolic or inflammatory variables. Because the archived creatinine field in the case dataset contained non-interpretable zero-coded values, the corrected analysis emphasizes markers that could be evaluated without introducing unverifiable assumptions.

2. MATERIALS AND METHODS

2.1 Study Design and Data Source

This retrospective analytical case-control study used two existing archived datasets (cases and controls). The analytical framework evaluated renal-related biochemical, metabolic, inflammatory, and atherogenic patterns in accordance with contemporary literature on chronic kidney disease and metabolic kidney vulnerability [1,6,10].

Data source and setting: The analysis was based on two archived spreadsheet datasets supplied for statistical analysis, one containing cases and the other controls. No participants were prospectively recruited for this study.

2.2 Study Population and Variables

Study population: The archived datasets contained 379 records in total, consisting of 173 cases and 206 controls. Variable-specific analyses used the observations available for the relevant variables rather than assuming complete data for all 379 records.

Recorded variables: Available variables included age, sex, disease onset and treatment information in the case dataset, together with biochemical measurements. Demographic comparisons in Table 1 were based on 49 cases and 49 controls with available age/sex data, while treatment percentages were based on 5 case records with documented therapy.

Renal-related biochemical markers: Sodium, potassium, chloride, and magnesium were evaluated as renal-related electrolyte markers. Creatinine was present in the archived files, but the case-side field consisted of zero-coded entries that could not be verified as physiological measurements and was therefore excluded from substantive inferential interpretation.

Metabolic markers: Insulin and HOMA-IR were treated as the core metabolic markers available in both study groups.

Inflammatory marker: hsCRP was used as the principal inflammatory indicator.

Atherogenic and lipid markers: Total cholesterol, HDL-C, triglycerides, LDL-C, sdLDL-C, non-HDL-C, TG/LDL-C, TG/HDL-C, non-HDL/HDL, non-HDL/LDL, and TC/TG were analyzed as the lipid and atherogenic domain [4,7,10-12].

2.3 Inclusion Criteria, Data Cleaning and Missing Data

Inclusion criteria: Records were eligible when group classification and at least one variable from the renal-related biochemical, metabolic, inflammatory, or lipid domains were available. Analyses were performed using variable-specific available data, and denominators therefore differ across tables.

Data cleaning and missing data: Clearly non-interpretable entries, duplicate rows, and blank records were excluded. The zero-coded creatinine values in the case dataset were treated as a data-quality limitation and were not replaced by generated values. Creatinine-derived inferential results were excluded from the corrected interpretation. Pairwise complete observations were used for correlation analyses.

2.4 Outcome Framework

Outcome framework: The study did not claim direct histopathologic renal injury or a clinical diagnosis of chronic kidney disease. Instead, it described a biochemical case-control pattern involving renal-related electrolyte, metabolic, inflammatory, and atherogenic domains [1].

2.5 Statistical Analysis

Data management and statistical analyses were performed using IBM SPSS Statistics version 31.0.2.0, and graphical outputs were prepared using Python 3.11. Continuous variables were summarized as mean ± standard deviation when approximately normally distributed and as median with interquartile range when non-normally distributed. Normality was assessed before inferential comparisons. Between-group comparisons used the independent-samples t-test with Welch correction or the Mann-Whitney U test, as appropriate. Categorical variables were compared using the chi-square test. Spearman rank correlation coefficients were calculated using pairwise complete observations to examine associations between renal-related electrolyte markers and metabolic or inflammatory parameters. A multivariable model was not retained in the corrected analysis because the archived creatinine field contained non-interpretable zero-coded case values and no source data were available to verify or reconstruct them. All tests were two-sided, and $p < 0.05$ was considered statistically significant.

3. RESULTS AND DISCUSSION

3.1 Demographic and Clinical Characteristics

Table 1. Demographic and clinical characteristics among records with available data

Characteristic	Cases	Controls	Test	p-value
Age (years)	53.00 (49.00-60.00)	31.00 (26.00-35.00)	Mann-Whitney U	<0.0001
Sex: M	15 (30.6%)	42 (85.7%)	Chi-square test	<0.0001
Sex: F	34 (69.4%)	7 (14.3%)		
Onset (cases only)	49.00 (42.00-54.00)			
Treatment (cases): MET./GLIB.	3 (60.0%)			
Treatment (cases): //	1 (20.0%)			
Treatment (cases): Daonil	1 (20.0%)			

Note: Age and sex comparisons are based on 49 cases and 49 controls with available demographic data. Treatment percentages are based on 5 case records with documented therapy. Percentages therefore use variable-specific denominators.

Among records with available demographic data, cases were older than controls and sex distribution differed significantly between groups. These imbalances are important when interpreting downstream biochemical comparisons. Treatment information was available for only five case records and is therefore presented descriptively rather than as a characterization of the full case dataset.

3.2 Descriptive Biochemical Profile

Table 2. Descriptive statistics of metabolic, inflammatory, atherogenic, and renal-related biochemical markers

Variable	Cases	Controls	Total
Na (mmol/L)	129.00 (126.00-134.00)	137.00 (118.00-143.00)	131.30 ± 13.70
K (mmol/L)	4.01 ± 0.59	4.60 (4.20-4.80)	4.20 (3.90-4.60)

Variable	Cases	Controls	Total
CL(mmol/L)	103.00 (100.00-106.00)	102.00 (97.00-108.00)	103.00 (99.25-107.00)
Mg(mmol/L)	0.80 (0.70-0.90)	0.80 (0.70-0.90)	0.80 (0.70-0.90)
insulin(μIU/mL)	6.40 (4.70-8.00)	5.50 (4.10-7.40)	5.50 (4.53-7.40)
HOMA-IR	2.55 (1.80-3.82)	1.20 (1.00-1.60)	1.70 (1.20-2.80)
hsCRP(mg/L)	6.30 (2.70-11.90)	5.90 (2.50-11.10)	5.90 (2.62-11.10)
TOTAL CHOL(mmol/L)	4.65 ± 1.28	3.20 (2.20-4.00)	3.95 (2.90-5.30)
HDL-C(mmol/L)	1.17 ± 0.60	1.33 (1.08-1.65)	1.27 (0.90-1.65)
TRIGLYC(mmol/L)	1.37 (0.87-1.82)	0.72 (0.43-1.39)	0.99 (0.61-1.54)
LDL-C(mmol/L)	4.30 ± 1.12	1.52 (1.09-2.14)	3.26 (1.52-4.64)
sdLDL-C	9.16 ± 0.38	10.08 (9.71-10.37)	9.51 ± 0.73
Non-HDL-C	3.48 ± 1.27	1.67 (1.16-2.43)	2.54 (1.65-3.85)
TG/LDL-C	0.30 (0.25-0.41)	0.39 (0.24-0.84)	0.32 (0.24-0.58)
TG/HDL-C	1.49 (0.78-2.22)	0.48 (0.35-0.90)	0.83 (0.43-1.67)
Non-HDL/HDL	2.86 (1.70-5.64)	1.31 (0.90-2.27)	2.00 (1.12-3.28)
Non-HDL/LDL	0.84 (0.68-0.89)	1.07 (1.04-1.14)	0.96 (0.80-1.07)
TC/TG	3.51 (2.64-4.39)	5.37 (2.34-6.82)	3.99 (2.51-5.49)

Note: Descriptive statistics are based on available observations and denominators vary across variables. FBS and creatinine were not retained in this summary because case-side availability/interpretability was insufficient for a valid pooled comparison.

Table 2 summarizes the available biochemical architecture of the study. The pattern is multidimensional and includes renal-related electrolytes, insulin-resistance indices, inflammatory activity, and atherogenic lipid burden. Several metabolic and lipid-derived variables showed wide dispersion, supporting the use of distribution-appropriate nonparametric comparisons where required. The descriptive results provide the basis for the subsequent between-group and correlation analyses without implying that every variable was available for every participant.

3.3 Renal-Related Electrolyte Markers

Table 3. Comparison of renal-related electrolyte markers between study groups

Variable	Cases	Controls	Test	p-value
Na (mmol/L)	129.00 (126.00-134.00)	137.00 (118.00-143.00)	Mann-Whitney U	0.6464
K (mmol/L)	4.00 (3.70-4.30)	4.60 (4.20-4.80)	Mann-Whitney U	<0.0001
CL(mmol/L)	103.00 (100.00-106.00)	102.00 (97.00-108.00)	Mann-Whitney U	0.6794
Mg(mmol/L)	0.80 (0.70-0.90)	0.80 (0.70-0.90)	Mann-Whitney U	0.5850

Note: Creatinine was excluded from inferential interpretation because the archived case-side field consisted of non-interpretable zero-coded values.

Table 3 shows a selective rather than generalized renal-related electrolyte pattern. Potassium differed significantly between groups, whereas sodium, chloride, and magnesium did not. This indicates that the available electrolyte data do not support a uniform renal disturbance across all measured markers. Creatinine was not used to support clinical or inferential conclusions because its case-side archived values could not be verified as physiological measurements.

3.4 Metabolic Parameters

Table 4. Comparison of metabolic parameters between study groups

Variable	Cases	Controls	Test	p-value
insulin(μIU/mL)	6.40 (4.70-8.00)	5.50 (4.10-7.40)	Mann-Whitney U	0.1267
HOMA-IR	2.55 (1.80-3.82)	1.20 (1.00-1.60)	Mann-Whitney U	<0.0001

Table 4 shows that HOMA-IR clearly differentiated the groups, whereas insulin alone did not reach statistical significance. This pattern suggests that the composite index of insulin resistance captured the metabolic contrast more effectively than insulin concentration in isolation. Accordingly, insulin resistance appears to be an important component of the observed biochemical case-control pattern.

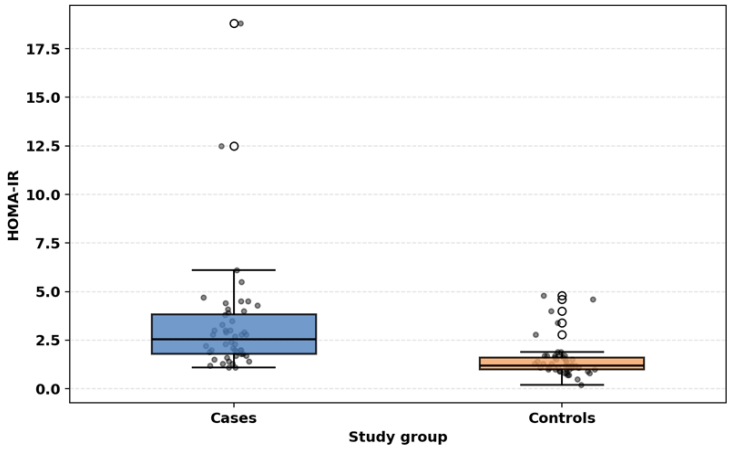

Figure 1. Distribution of HOMA-IR by study groups.

3.5 Inflammatory and Atherogenic Indices

Table 5. Comparison of inflammatory and atherogenic indices between study groups

Variable	Cases	Controls	Test	p-value
hsCRP(mg/L)	6.30 (2.70-11.90)	5.90 (2.50-11.10)	Mann-Whitney U	0.8869
TOTAL CHOL(mmol/L)	4.70 (3.80-5.40)	3.20 (2.20-4.00)	Mann-Whitney U	0.0002
HDL-C(mmol/L)	1.15 (0.61-1.62)	1.33 (1.08-1.65)	Mann-Whitney U	0.0977
TRIGLYC(mmol/L)	1.37 (0.87-1.82)	0.72 (0.43-1.39)	Mann-Whitney U	<0.0001
LDL-C(mmol/L)	4.30 (3.54-4.99)	1.52 (1.09-2.14)	Mann-Whitney U	<0.0001
sdLDL-C	9.23 (8.85-9.43)	10.08 (9.71-10.37)	Mann-Whitney U	<0.0001

Variable	Cases	Controls	Test	p-value
Non-HDL-C	3.36 (2.58-4.09)	1.67 (1.16-2.43)	Mann-Whitney U	<0.0001
TG/LDL-C	0.30 (0.25-0.41)	0.39 (0.24-0.84)	Mann-Whitney U	0.0793
TG/HDL-C	1.49 (0.78-2.22)	0.48 (0.35-0.90)	Mann-Whitney U	<0.0001
Non-HDL/HDL	2.86 (1.70-5.64)	1.31 (0.90-2.27)	Mann-Whitney U	<0.0001
Non-HDL/LDL	0.84 (0.68-0.89)	1.07 (1.04-1.14)	Mann-Whitney U	<0.0001
TC/TG	3.51 (2.64-4.39)	5.37 (2.34-6.82)	Mann-Whitney U	0.0068

Table 5 shows the broadest between-group separation in the lipid and atherogenic domain. Several conventional lipid variables and derived ratios differed significantly, indicating that dyslipidemic burden was a prominent feature of the case-control pattern. In contrast, hsCRP was not a significant discriminator. These results suggest a stronger metabolic and atherogenic signal than an hsCRP-based inflammatory signal in the available sample.

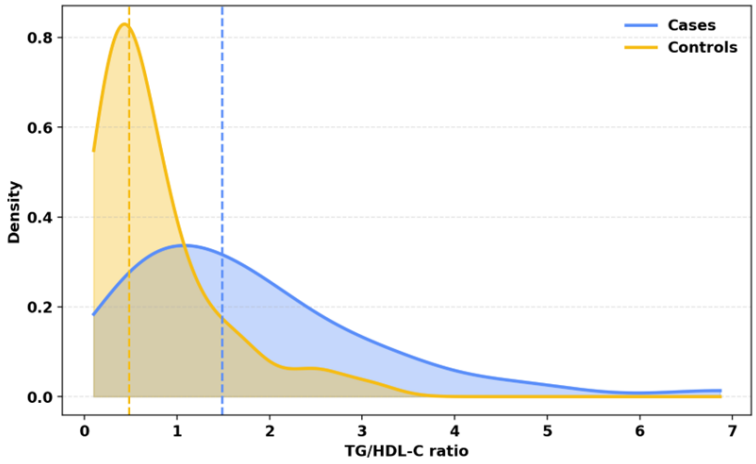

Figure 2. Distribution of TG/HDL-C ratio by study groups.

3.6 Correlation Analyses

Table 6. Correlation between renal-related electrolyte markers and metabolic parameters (Spearman)

Renal Marker	Metabolic Marker	N	Spearman rho	p-value
Na (mmol/L)	insulin(μIU/mL)	98	-0.085	0.4027
Na (mmol/L)	HOMA-IR	97	-0.046	0.6563
K (mmol/L)	insulin(μIU/mL)	98	-0.268	0.0077
K (mmol/L)	HOMA-IR	97	-0.379	0.0001
CL(mmol/L)	insulin(μIU/mL)	98	-0.189	0.0624
CL(mmol/L)	HOMA-IR	97	-0.034	0.7416

Renal Marker	Metabolic Marker	N	Spearman rho	p-value
Mg(mmol/L)	insulin(μIU/mL)	98	-0.160	0.1161
Mg(mmol/L)	HOMA-IR	97	-0.121	0.2362

Table 6 shows that potassium was significantly associated with both insulin and HOMA-IR, whereas the other evaluated electrolyte markers did not show statistically significant metabolic correlations. The association pattern was therefore selective and concentrated mainly in potassium rather than being distributed across all renal-related biochemical markers.

Table 7. Correlation between renal-related electrolyte markers and hsCRP (Spearman)

Renal Marker	Inflammatory Marker	N	Spearman rho	p-value
Na (mmol/L)	hsCRP(mg/L)	98	-0.038	0.7089
K (mmol/L)	hsCRP(mg/L)	98	0.193	0.0570
CL(mmol/L)	hsCRP(mg/L)	98	0.124	0.2240
Mg(mmol/L)	hsCRP(mg/L)	98	0.080	0.4320

None of the correlations between the evaluated renal-related electrolyte markers and hsCRP reached statistical significance (Table 7). This is consistent with the non-significant between-group comparison for hsCRP and indicates that hsCRP was not a dominant statistical signal in the present dataset. The finding does not exclude a biological role for inflammation in kidney disease; it only describes the pattern observed in these archived data.

3.7 Integrated Discussion and Study Limitations

The current results suggest that the biochemical case-control pattern was selective and multidimensional rather than uniformly distributed across all measured variables. Among the renal-related electrolyte markers, potassium showed a clear between-group difference, while sodium, chloride, and magnesium were comparatively stable. Because the archived case-side creatinine values were zero-coded and could not be verified, they were not interpreted as physiological measurements. This data-quality limitation necessarily restricts renal-specific conclusions.

The metabolic findings support a distinct role for insulin resistance. The groups differed in HOMA-IR but not in insulin alone, indicating that the composite insulin-resistance index captured the metabolic contrast more clearly. This is consistent with evidence linking insulin resistance to endothelial dysfunction, oxidative stress, altered sodium handling, and microvascular injury [6], as well as cohort and retrospective studies associating insulin-resistance indices with chronic kidney disease phenotypes [5,9].

The most widespread differences were observed in the atherogenic and lipid-derived markers. Triglycerides, LDL-C, non-HDL-C, TG/HDL-C, and related ratios showed strong case-control contrasts, indicating that dyslipidemic burden was a major component of the biochemical pattern. This finding is consistent with studies linking elevated TG/HDL-C and broader atherogenic profiles with renal-function decline and advanced CKD [4,12], and with reviews describing dyslipidemia as a biologically active process in CKD [7,10].

hsCRP did not differ significantly between groups and did not show significant correlations with the evaluated renal-related electrolyte markers. This does not exclude inflammation from kidney disease biology, but it indicates that hsCRP was not the dominant inflammatory signal in this archived sample. Previous studies have associated persistent hsCRP elevation with poorer

renal outcomes in metabolically vulnerable populations [3], while mechanistic work has implicated CRP in renal inflammatory and fibrotic pathways [2].

Correlation analysis further refined the pattern: potassium was associated with insulin and HOMA-IR, whereas the remaining electrolyte markers showed weaker or non-significant metabolic relationships, and no evaluated electrolyte marker was significantly correlated with hsCRP. These findings support a selective relationship between metabolic disturbance and specific renal-related biochemical markers rather than a generalized systemic effect across all measured variables.

Overall, the available data support a multidomain biochemical pattern in which insulin resistance and atherogenic dyslipidemia were more prominent than hsCRP-based inflammation, with potassium providing the clearest renal-related electrolyte signal. The findings are best interpreted as exploratory biochemical associations rather than as evidence of established renal injury, because direct renal indices such as eGFR and albuminuria were unavailable and the archived creatinine field could not be clinically interpreted.

A strength of the present study is the simultaneous assessment of metabolic, inflammatory, atherogenic, and renal-related biochemical domains in a single analytical framework, including several derived lipid indices. Important limitations must also be recognized. The case and control groups were not evenly matched for age and sex, demographic and treatment information was available only for subsets of records, and variable-specific missingness produced different denominators across analyses. The archived case-side creatinine field contained zero-coded values that could not be verified as physiological measurements and was therefore excluded from substantive inferential interpretation. In addition, eGFR, albuminuria, urinary biomarkers, and histopathologic confirmation were unavailable. The findings should therefore be viewed as an exploratory biochemical case-control characterization rather than a diagnostic or mechanistic renal study.

4. CONCLUSION

This analysis identifies a multidimensional biochemical case-control pattern characterized by significant HOMA-IR and atherogenic lipid differences, a selective potassium signal, and comparatively limited hsCRP contribution. The results support further investigation of metabolic and atherogenic abnormalities alongside validated renal measures. Future studies should incorporate verified creatinine measurements, eGFR, albuminuria, and urinary biomarkers; improve demographic matching and completeness of clinical data; and externally validate the observed lipid-derived associations in independent samples.

ACKNOWLEDGEMENTS

None.

AUTHOR CONTRIBUTIONS

Saif Salah Abdul Hassan was responsible for study conceptualization, statistical analysis, figure preparation, interpretation of results, and manuscript drafting.

CONFLICT OF INTEREST

The author declares that there is no conflict of interest related to this work.

FUNDING

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

ETHICS STATEMENT

This study analyzed previously archived, de-identified secondary datasets. No new participant recruitment or biological sample collection was undertaken for the present analysis.

DATA AVAILABILITY STATEMENT

The de-identified processed analytical dataset and analysis outputs supporting the findings are available from the corresponding author upon reasonable academic request, subject to applicable confidentiality and data-use restrictions.

REFERENCES

[1] Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2024. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney International 105(4 Suppl), S117-S314. https://doi.org/10.1016/S0085-2538(24)00110-8.

[2] Li, J., Chen, J., Lan, H.Y., Tang, Y., 2023. Role of C-Reactive Protein in Kidney Diseases. Kidney Diseases 9(2), 73-81. https://doi.org/10.1159/000528693.

[3] Liu, L., Gao, B., Wang, J., et al., 2021. Clinical significance of single and persistent elevation of serum high-sensitivity C-reactive protein levels for prediction of kidney outcomes in patients with impaired fasting glucose or diabetes mellitus. Journal of Nephrology 34(4), 1179-1188. https://doi.org/10.1007/s40620-020-00848-4.

[4] Lv, S., Zhang, H., Chen, J., et al., 2021. The effect of triglycerides to high-density lipoprotein cholesterol ratio on the reduction of renal function: findings from China Health and Retirement Longitudinal Study (CHARLS). Lipids in Health and Disease 20, 110. https://doi.org/10.1186/s12944-021-01542-5.

[5] Mu, X., Wu, A., Hu, H., Yang, M., Zhou, H., 2024. Correlation between alternative insulin resistance indexes and diabetic kidney disease: a retrospective study. Endocrine 84(1), 136-147. https://doi.org/10.1007/s12020-023-03574-6.

[6] Nakashima, A., Kato, K., Ohkido, I., Yokoo, T., 2021. Role and Treatment of Insulin Resistance in Patients with Chronic Kidney Disease: A Review. Nutrients 13(12), 4349. https://doi.org/10.3390/nu13124349.

[7] Russo, G., Piscitelli, P., Giandalia, A., Viazzi, F., Pontremoli, R., Fioretto, P., et al., 2020. Atherogenic dyslipidemia and diabetic nephropathy. Journal of Nephrology 33(5), 1001-1008. https://doi.org/10.1007/s40620-020-00739-8.

[8] Shen, R., Lin, L., Bin, Z., Qiao, X., 2024. The U-shape relationship between insulin resistance-related indexes and chronic kidney disease: a retrospective cohort study from National Health and Nutrition Examination Survey 2007-2016. Diabetology & Metabolic Syndrome 16, 168. https://doi.org/10.1186/s13098-024-01408-7.

[9] Song, S.H., Goo, Y.J., Oh, T.R., Suh, S.H., Choi, H.S., Kim, C.S., et al., 2025. Insulin resistance is associated with incident chronic kidney disease in population with normal renal function. Kidney Research and Clinical Practice 44(3), 491-499. https://doi.org/10.23876/j.krcp.22.298.

[10] Suh, S.H., Kim, S.W., 2023. Dyslipidemia in Patients with Chronic Kidney Disease: An Updated Overview. Diabetes & Metabolism Journal 47(5), 612-629. https://doi.org/10.4093/dmj.2023.0067.

[11] Suh, S.H., Oh, T.R., Choi, H.S., et al., 2022. Non-High-Density Lipoprotein Cholesterol and Cardiovascular Outcomes in Chronic Kidney Disease: Results from KNOW-CKD Study. Nutrients 14(18), 3792. https://doi.org/10.3390/nu14183792.

[12] Weldegiorgis, M., Woodward, M., 2022. Elevated triglycerides and reduced high-density lipoprotein cholesterol are independently associated with the onset of advanced chronic kidney disease: a cohort study of 911,360 individuals from the United Kingdom. BMC Nephrology 23, 312. https://doi.org/10.1186/s12882-022-02932-2.