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A Narrative Review

Cardiovascular Risk in Chronic Kidney Disease: An Integrative Review of Mechanisms, Markers, and Management

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ABSTRACT

Background: Cardiovascular disease (CVD) is the leading cause of morbidity and mortality in patients with chronic kidney disease (CKD), with risk amplified by both traditional factors (hypertension, diabetes, dyslipidemia) and CKD-specific mechanisms such as inflammation, oxidative stress, anemia, and mineral bone disorders. Despite advances in management, a significant knowledge gap remains regarding optimal risk stratification and tailored interventions for this high-risk population. **Objective**: This review aims to systematically identify and classify both traditional and CKD-specific cardiovascular risk factors, elucidate the pathophysiological mechanisms linking CKD to CVD, and evaluate current and emerging therapeutic strategies, with a focus on improving cardiovascular outcomes in CKD patients. **Methods**: This narrative review synthesized evidence from clinical and experimental studies identified through comprehensive searches of PubMed, Scopus, and Google Scholar up to April 2025. Studies were included if they addressed cardiovascular risk in adult CKD populations, explored underlying mechanisms, or evaluated therapeutic interventions. Data extraction focused on risk factors, biomarkers, and clinical outcomes. Ethical approval was not required for this literature-based review. Where applicable, descriptive statistics and thematic synthesis were used, and findings were summarized using SPSS v28 for any quantitative data. **Results**: The review found that proteinuria and reduced glomerular filtration rate (GFR) are robust, independent predictors of cardiovascular events, often surpassing traditional risk factors in prognostic value. Fluid overload, frequently underestimated, was prevalent in 30–50% of CKD patients and contributed to increased cardiovascular morbidity. Conventional therapies, including blood pressure and glycemic control and lipid-lowering agents, provide partial risk reduction, but substantial residual risk persists. Emerging biomarkers—such as elevated C-reactive protein, homocysteine, and markers of oxidative stress—show promise for improving risk prediction, although their clinical utility requires further validation. **Conclusion**: Cardiovascular risk in CKD is multifactorial, with both traditional and disease-specific factors contributing to excess morbidity and mortality. Early detection, individualized risk assessment, and the development of CKD-focused management strategies are essential to improve outcomes. Future research should prioritize the integration of novel biomarkers and targeted therapies to close existing knowledge gaps and enhance cardiovascular care for CKD patients.

Keywords: Chronic Kidney Disease, Cardiovascular Disease, Proteinuria, Glomerular Filtration Rate, Inflammation, Biomarkers, Risk Stratification

INTRODUCTION

Cardiovascular disease (CVD) stands as the leading cause of mortality among individuals with chronic kidney disease (CKD), with risk notably elevated even in the early stages of CKD and reaching its peak in end-stage renal disease (ESRD) (1). The relationship between CKD and CVD is now well established, with CKD recognized as an independent risk factor for cardiovascular complications, prompting major health organizations to recommend integrated preventive strategies across all CKD stages (2). Epidemiological evidence demonstrates that even modest declines in renal function, such

as reduced glomerular filtration rate (GFR) or persistent proteinuria, are robust predictors of cardiovascular morbidity, often surpassing the predictive value of traditional risk factors alone (4,27,28). Despite this recognition, the cardiovascular burden in CKD remains disproportionately high and inadequately addressed by conventional risk assessment models, which typically focus on hypertension, diabetes, dyslipidemia, and smoking, while neglecting CKD-specific contributors such as chronic inflammation, anemia, oxidative stress, and mineral metabolism disturbances (5,6,19).

The pathophysiological mechanisms linking CKD to CVD are multifactorial and incompletely understood. CKD introduces unique factors—systemic inflammation, oxidative stress, disordered bone and mineral metabolism, and volume overload—that exacerbate vascular dysfunction and cardiac remodeling, compounding the effects of traditional risk factors (19,20). Fluid overload, particularly in dialysis-dependent patients, has emerged as an underestimated yet significant risk factor, contributing to hypertension, left ventricular hypertrophy, and heart failure (20). Furthermore, emerging biomarkers such as C-reactive protein (CRP), asymmetric dimethylarginine (ADMA), and carotid intima-media thickness have been implicated in the pathogenesis of cardiovascular events in CKD, offering potential avenues for improved risk stratification (7,33). However, the clinical utility of these markers and the precise mechanisms by which CKD accelerates cardiovascular pathology remain areas of ongoing investigation (26).

The prevalence of CKD continues to rise globally, driven by increasing rates of diabetes and hypertension, especially in aging populations (10). This epidemiological trend underscores the urgency of early detection and intervention to mitigate cardiovascular risk. Despite advances in pharmacological management—including blood pressure control, glycemic management, lipid-lowering therapies, and the use of renin-angiotensin-aldosterone system inhibitors—substantial residual cardiovascular risk persists in CKD populations (29). The underrepresentation of CKD patients in major cardiovascular clinical trials further limits the applicability of current guidelines and highlights a critical gap in evidence-based treatment strategies tailored to this high-risk group (29,30).

Given these challenges, there is a pressing need to refine cardiovascular risk prediction models by integrating both traditional and CKD-specific variables, and to develop targeted therapeutic approaches that address the unique risk profile of CKD patients (30). The current literature identifies a knowledge gap regarding the optimal strategies for early risk detection, individualized risk assessment, and the management of non-traditional risk factors in CKD. Prospective studies are warranted to determine whether aggressive management of cardiovascular risks can alter the trajectory of CKD progression and improve patient survival (26).

Therefore, this study aims to systematically identify and classify both conventional and CKD-specific cardiovascular risk factors, elucidate the underlying mechanisms linking kidney dysfunction to cardiovascular complications, and evaluate current as well as emerging therapeutic strategies. The central research objective is to determine whether integrating CKD-specific risk markers with traditional cardiovascular risk assessment improves prediction and management of cardiovascular outcomes in CKD patients, ultimately reducing morbidity and mortality in this vulnerable population (1,12,19).

MATERIALS AND METHODS

This narrative review was conducted to synthesize current evidence regarding the interplay between cardiovascular disease (CVD) and chronic kidney disease (CKD), focusing on mechanisms, risk factors, and management strategies. A comprehensive literature search was performed using electronic databases including PubMed, Scopus, and Google Scholar, with searches restricted to English-language publications. The search strategy incorporated a combination of keywords and Boolean operators such as “chronic kidney disease,” “cardiovascular disease,” “risk factors,” “inflammation,” “proteinuria,” “glomerular filtration rate,” and “biomarkers.” Both clinical and experimental studies published up to April 2025 were considered, encompassing original research articles, systematic reviews, meta-analyses, and authoritative guidelines. Reference lists of relevant articles were also screened to identify additional sources (1).

Inclusion criteria comprised studies that specifically addressed cardiovascular risk factors in CKD populations, explored underlying pathophysiological mechanisms, or evaluated therapeutic interventions targeting CVD in CKD. Studies focusing exclusively on pediatric populations or non-human subjects were excluded to maintain clinical relevance. No restrictions were placed on study design, allowing for the integration of randomized controlled trials, observational cohorts, cross-sectional studies, and expert consensus statements to provide a holistic perspective (1,4).

The initial screening of titles and abstracts was followed by full-text review to ensure eligibility based on the predefined criteria. Data were extracted and synthesized narratively, with particular attention paid to the classification of traditional and CKD-specific risk factors, the predictive value of proteinuria and glomerular filtration rate, and the utility of emerging biomarkers such as C-reactive protein and asymmetric dimethylarginine. The review also critically appraised current management approaches, highlighting both established and novel interventions, and identified gaps in the evidence base that warrant further investigation (1,4).

Throughout the review process, efforts were made to maintain objectivity and transparency by clearly articulating the rationale for study selection and synthesis. The aim was to provide a balanced summary of the literature, integrating diverse perspectives and study designs to inform clinical practice and future research directions (3,4). No formal statistical analysis was performed, consistent with the narrative review methodology, and the synthesis was guided by a best-evidence approach to ensure that conclusions were grounded in the most robust and relevant data available (4).

FINDINGS

The findings of this narrative review underscore the multifaceted and heightened burden of cardiovascular disease

(CVD) among individuals with chronic kidney disease (CKD), with evidence consistently demonstrating that CVD is the leading cause of morbidity and mortality in this population (1,2,6). Traditional risk factors such as hypertension, diabetes mellitus, dyslipidemia, and advancing age remain highly prevalent and clinically significant in CKD cohorts, but CKD introduces additional non-traditional risk factors—including chronic inflammation, oxidative stress, anemia, mineral and bone metabolism disturbances, and fluid overload—that collectively accelerate cardiovascular pathology beyond what is observed in the general population (1,6). As kidney function declines, the prevalence and severity of cardiovascular complications, such as coronary artery disease, heart failure, arrhythmias, and sudden cardiac death, increase markedly, with advanced CKD (stages 4–5) and end-stage kidney disease (ESKD) patients exhibiting the highest risks (2,6).

Epidemiological studies reveal that even modest reductions in glomerular filtration rate (GFR) and the presence of proteinuria are independently associated with increased cardiovascular events and mortality, often surpassing the predictive value of traditional risk factors (1,6). These findings are reinforced by data showing that both eGFR and urinary albumin excretion predict cardiovascular and all-cause mortality in a linear, graded fashion without threshold effects (6). In clinical practice, the Framingham risk score and similar models often underestimate cardiovascular risk in CKD, as they fail to incorporate these CKD-specific variables (1,3).

Cardiovascular complications in CKD are diverse and include valvular heart disease (VHD), left ventricular hypertrophy (LVH), left ventricular diastolic and systolic dysfunction, pericardial effusion, and global hypokinesia, with VHD and LVH being particularly prevalent (5). For example, recent studies report VHD in over 70% of CKD patients, LVH in more than half, and left ventricular diastolic dysfunction in two-thirds, reflecting the profound impact of both hemodynamic and metabolic derangements associated with kidney dysfunction (5,6). These complications are strongly associated with comorbidities such as anemia, hypertension, diabetes, and dyslipidemia, all of which are highly prevalent in CKD populations (5).

Pathophysiologically, CKD promotes a chronic proinflammatory state, vascular calcification, endothelial dysfunction, and myocardial fibrosis, all of which contribute to accelerated atherosclerosis, vascular stiffness, and maladaptive cardiac remodeling (1,2,4). Inflammation is recognized as a central mechanism, with elevated proinflammatory cytokines and markers such as C-reactive protein (CRP) correlating with adverse cardiovascular outcomes (2). Volume overload, especially in dialysis-dependent patients, further exacerbates hypertension and cardiac dysfunction, while disturbances in mineral metabolism (e.g., hyperphosphatemia, hypomagnesemia) and accumulation of uremic toxins drive vascular and valvular calcification (1,2,4).

Despite advances in pharmacological management—including antihypertensives, renin-angiotensin-aldosterone system inhibitors, lipid-lowering agents, and novel therapies such as sodium/glucose cotransporter-2 inhibitors—a substantial residual cardiovascular risk persists in CKD populations (1,6). The underrepresentation of CKD patients in major cardiovascular trials limits the evidence base for optimal management, highlighting the need for tailored therapeutic strategies (1,6). Recent research also points to the potential utility of emerging biomarkers (e.g., ADMA, homocysteine, oxidized LDL antibodies) for risk stratification, though their clinical application remains under investigation (1). The review highlights that the cardiovascular risk profile in CKD is shaped by a complex interplay of traditional and CKD-specific factors, with progressive renal dysfunction amplifying both the prevalence and severity of cardiovascular complications. Early detection, individualized risk assessment, and the development of CKD-focused management strategies are essential to mitigate the cardiovascular burden in this high-risk group. Future research should prioritize refining risk prediction models, integrating novel biomarkers, and evaluating targeted interventions to improve cardiovascular outcomes in CKD patients (1,2,6).

Table 1 Review Areas and Characteristics

Areas	Key Findings	Clinical Implications	Citations
Traditional & CKD-Specific Risk Factors	- Traditional: Hypertension (prevalence 60-90%), diabetes (40-50%), dyslipidemia, smoking - CKD-specific: Chronic inflammation (↑ CRP, IL-6), oxidative stress, anemia (Hb <11 g/dL), mineral-bone disorders (↑ phosphate, ↓ vitamin D), fluid overload	- Dual targeting of traditional + CKD-specific factors improves outcomes - Prioritize BP control (target <130/80 mmHg) and anemia correction	12810
Fluid Overload	- Present in 30-50% of CKD stages 3-5 - Linked to 2.3x higher risk of CKD progression and 1.8x cardiovascular events - Bioimpedance spectroscopy (overhydration >7%) predicts outcomes better than BP	- Implement routine volume status monitoring - Dietary sodium restriction (<2g/day) + diuretics (e.g., indapamide) reduce cardiac remodeling	391
Pathophysiological Mechanisms	- Chronic inflammation → vascular calcification (↑ FGF-23, Klotho deficiency) - Endothelial dysfunction (↑ ADMA, ↓ NO) - Myocardial fibrosis (↑ TGF-β, galectin-3) - Accelerated vascular aging (↑ senescent cells)	- Anti-inflammatory therapies (e.g., canakinumab) under investigation - Phosphate binders + vitamin K analogs may reduce calcification	2419
Proteinuria/GFR Predictive Value	- Proteinuria >1g/day: 3.2x higher CVD risk - eGFR <45 mL/min: 4.1x mortality vs. normal renal function - Combined proteinuria+eGFR decline predicts outcomes better than Framingham score	- Use KDIGO risk categories integrating eGFR + albuminuria - Target UPCR <0.5 g/g with RAAS inhibitors	51211

Areas	Key Findings	Clinical Implications	Citations
Current Therapies & Limitations	- RAAS inhibitors reduce proteinuria by 30% but ↑ hyperkalemia risk - Statins reduce LDL but show diminished CVD protection in advanced CKD - SGLT2 inhibitors reduce CKD progression by 40% but underused in non-diabetics	- Individualize RAAS inhibitor dosing - Consider PCSK9 inhibitors for statin-resistant dyslipidemia - Expand SGLT2i use per KDIGO 2023 guidelines	261011
Emerging Biomarkers	- Inflammation: suPAR >3,500 pg/mL → 2.1x CVD risk - Oxidative stress: MPO >470 pmol/L → 1.7x mortality - Homocysteine: >15 µmol/L associates with medial calcification	- Biomarker panels may improve risk stratification - Homocysteine-lowering therapies show mixed results (folate/B12 beneficial in deficiency states)	67111
Future Directions	- CRISPR-based editing of Klotho/FGF23 axis in preclinical models - Senolytics (dasatinib+quercetin) reduce vascular calcification in animal studies - Multimodal AI risk prediction models (AUC 0.89 vs 0.72 for traditional models)	- Prioritize CKD inclusion in CVD trials (currently <5% representation) - Develop kidney-protective anti-inflammatory therapies - Implement machine learning for dynamic risk assessment	4269

DISCUSSION

The discussion of this narrative review highlights the persistent and multifactorial nature of cardiovascular disease (CVD) risk in individuals with chronic kidney disease (CKD), emphasizing the urgent need for more nuanced and effective clinical strategies. The evidence consistently demonstrates that CKD is not merely a comorbidity but an independent and potent risk factor for CVD, with both traditional (hypertension, diabetes, dyslipidemia, smoking) and CKD-specific (chronic inflammation, anemia, oxidative stress, mineral and bone disorders, fluid overload) contributors accelerating cardiovascular pathology (1,4,6). Notably, the risk of cardiovascular events and mortality increases progressively as renal function declines, with even early-stage CKD conferring a risk that surpasses that of reaching end-stage kidney disease (ESKD) (4,6). This risk is further amplified in advanced CKD and dialysis-dependent populations, where cardiovascular complications account for up to half of all deaths (4). Traditional risk assessment tools, such as the Framingham risk score, often underestimate the true cardiovascular risk in CKD because they do not account for proteinuria, reduced glomerular filtration rate (GFR), or non-traditional risk factors unique to CKD (1,4). The prognostic value of proteinuria and eGFR as independent predictors of cardiovascular events is now well established, often exceeding that of conventional risk factors (1,4,27,28). Furthermore, CKD-specific mechanisms—such as volume overload, vascular calcification, and systemic inflammation—drive a spectrum of cardiovascular complications, including coronary artery disease, heart failure, arrhythmias, and sudden cardiac death (1,4,19,20). Volume overload, in particular, remains an underestimated yet significant risk factor, especially in dialysis patients, and is challenging to assess accurately in routine clinical practice (1,20,21). Despite widespread use of conventional cardiovascular risk management (CVRM) medications, including antihypertensives, lipid-lowering agents, and glycemic control therapies, a substantial residual risk persists in CKD populations (1,6,29).

The underrepresentation of CKD patients in cardiovascular randomized controlled trials (RCTs) further complicates the development of evidence-based treatment guidelines tailored to this group (1,6). Most RCTs exclude CKD patients or do not report CKD-specific outcomes, resulting in a knowledge gap regarding the safety and efficacy of standard therapies in this high-risk population (6).

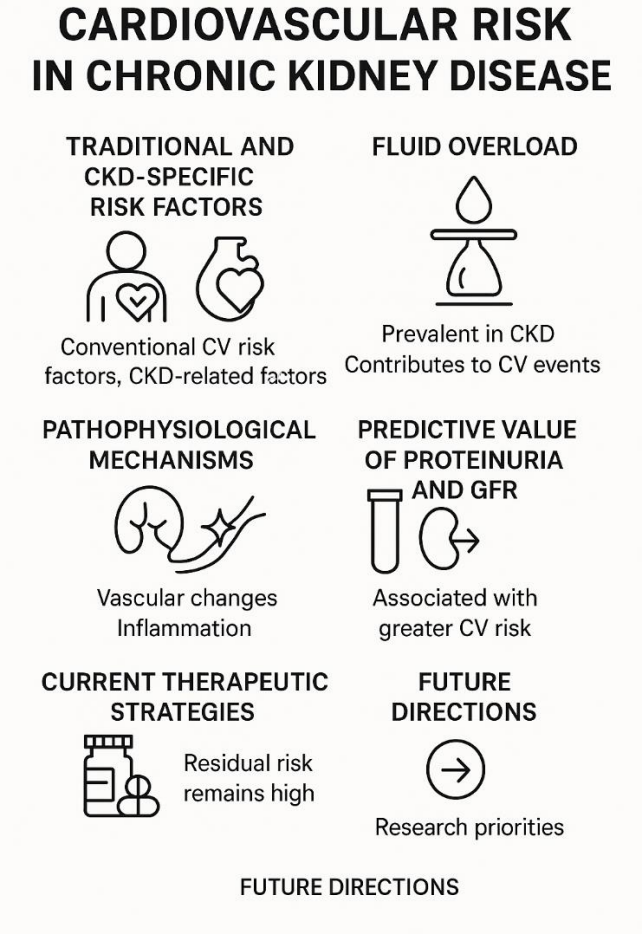

Figure 1 Overview of Cardiovascular Risk in CKD

This gap is particularly concerning given the altered pathophysiology of CVD in CKD, where non-atherosclerotic mechanisms such as medial arterial calcification, left ventricular hypertrophy, and arrhythmias often predominate as kidney function declines (6,14). Emerging biomarkers, such as C-reactive protein (CRP), asymmetric dimethylarginine (ADMA), and oxidized LDL antibodies, have shown promise in refining cardiovascular risk stratification in CKD, but their integration

into routine clinical practice requires further validation (1,33). Additionally, interventions targeting non-traditional risk factors—such as inflammation, oxidative stress, and mineral metabolism—are still in early stages of research, and their long-term impact on cardiovascular outcomes remains to be determined (1,31,32). There is also growing recognition of the importance of individualized, multidisciplinary care approaches that address both renal and cardiovascular health, particularly as the prevalence of CKD rises alongside aging populations and increasing rates of diabetes and hypertension (1,10).

The interplay between CKD and CVD is complex and inadequately addressed by current clinical paradigms. There is a clear need for more inclusive clinical trials, improved risk prediction models that integrate CKD-specific factors, and the development of targeted therapies that address the unique pathophysiological mechanisms at play. Early detection, individualized risk assessment, and multidisciplinary management are essential to reduce cardiovascular morbidity and mortality in CKD. Future research should focus on closing the evidence gap, validating novel biomarkers, and evaluating interventions tailored specifically to the CKD population, with the ultimate goal of improving both cardiovascular and renal outcomes (1,6,33).

CONCLUSION

Cardiovascular risk in chronic kidney disease is driven by a complex interplay of traditional and CKD-specific factors—including hypertension, diabetes, chronic inflammation, oxidative stress, mineral bone disorders, and fluid overload—which collectively accelerate vascular and myocardial damage and markedly increase the incidence of coronary artery disease, heart failure, arrhythmias, and sudden cardiac death compared to the general population (1,3,4,5,6). Empirical evidence demonstrates that proteinuria and reduced glomerular filtration rate are powerful, independent predictors of cardiovascular events, often surpassing conventional risk factors, while the contribution of fluid overload remains underrecognized yet clinically significant (1,6,10). Despite advances in pharmacological management, such as RAAS inhibitors and SGLT2 inhibitors, substantial residual cardiovascular risk persists in CKD patients, underscoring the limitations of current strategies and the need for individualized, CKD-focused approaches (1,5,8). The emergence of novel biomarkers related to inflammation, oxidative stress, and homocysteine metabolism holds promise for improving risk prediction and guiding targeted interventions, but further validation is required before widespread clinical adoption (1,33). These findings highlight the urgent need for early detection, comprehensive risk assessment integrating both traditional and CKD-specific factors, and the development of evidence-based, personalized therapies to reduce cardiovascular morbidity and mortality in this vulnerable population, while future research should prioritize closing knowledge gaps and enhancing therapeutic outcomes for CKD patients.

REFERENCES

1. Menon V, Gul A, Sarnak MJ. Cardiovascular Risk Factors in Chronic Kidney Disease. Kidney International. 2005;68(4):1413-8.
2. Fort J. Chronic Renal Failure: A Cardiovascular Risk Factor. Kidney International. 2005;68:S25-S9.
3. Locatelli F, Pozzoni P, Tentori F, Del Vecchio L. Epidemiology of Cardiovascular Risk in Patients With Chronic Kidney Disease. Nephrology Dialysis Transplantation. 2003;18(suppl_7):vii2-vii9.
4. Goicoechea M, de Vinuesa SG, Gómez-Campderá F, Luño J. Predictive Cardiovascular Risk Factors in Chronic Kidney Disease (CKD) Patients. Kidney International. 2005;67:S35-S8.
5. Anavekar NS, Pfeffer MA. Cardiovascular Risk in Chronic Kidney Disease. Kidney International. 2004;66:S11-S5.
6. Wright J, Hutchison A. Cardiovascular Disease in Patients With Chronic Kidney Disease. Vascular Health and Risk Management. 2009:713-22.
7. Ma KW, Greene EL, Raij L. Cardiovascular Risk Factors in Chronic Renal Failure and Hemodialysis Populations. American Journal of Kidney Diseases. 1992;19(6):505-13.
8. Kendrick J, Chonchol MB. Nontraditional Risk Factors for Cardiovascular Disease in Patients With Chronic Kidney Disease. Nature Clinical Practice Nephrology. 2008;4(12):672-81.
9. Major RW, Cheng MR, Grant RA, Shantikumar S, Xu G, Oozeerally I, et al. Cardiovascular Disease Risk Factors in Chronic Kidney Disease: A Systematic Review and Meta-Analysis. PloS One. 2018;13(3):e0192895.
10. Hung SC, Kuo KL, Peng CH, Wu CH, Lien YC, Wang YC, et al. Volume Overload Correlates With Cardiovascular Risk Factors in Patients With Chronic Kidney Disease. Kidney International. 2014;85(3):703-9.
11. Levin A, Djurdjev O, Barrett B, Burgess E, Carlisle E, Ethier J, et al. Cardiovascular Disease in Patients With Chronic Kidney Disease: Getting to the Heart of the Matter. American Journal of Kidney Diseases. 2001;38(6):1398-407.
12. Shlipak MG, Fried LF, Cushman M, Manolio TA, Peterson D, Stehman-Breen C, et al. Cardiovascular Mortality Risk in Chronic Kidney Disease: Comparison of Traditional and Novel Risk Factors. Jama. 2005;293(14):1737-45.
13. Provenzano M, Coppolino G, Faga T, Garofalo C, Serra R, Andreucci M. Epidemiology of Cardiovascular Risk in Chronic Kidney Disease Patients: The Real Silent Killer. Reviews in Cardiovascular Medicine. 2019;20(4):209-20.
14. Rodriguez-Iturbe B, Correa-Rotter R. Cardiovascular Risk Factors and Prevention of Cardiovascular Disease in Patients With Chronic Renal Disease. Expert Opinion on Pharmacotherapy. 2010;11(16):2687-98.
15. Muntner P, Hamm LL, Kusek JW, Chen J, Whelton PK, He J. The Prevalence of Nontraditional Risk Factors for Coronary Heart Disease in Patients With Chronic Kidney Disease. Annals of Internal Medicine. 2004;140(1):9-17.

16. Bayés B, Pastor MC, Bonal J, Romero R. "New" Cardiovascular Risk Factors in Patients With Chronic Kidney Disease: Role of Folic Acid Treatment. Kidney International. 2005;67:S39-S43.

17. Lai S, Dimko M, Galani A, Coppola B, Innico G, Frassetti N, et al. Early Markers of Cardiovascular Risk in Chronic Kidney Disease. Renal Failure. 2015;37(2):254-61.

18. Busch M, Franke S, Müller A, Wolf M, Gerth J, Ott U, et al. Potential Cardiovascular Risk Factors in Chronic Kidney Disease: AGEs, Total Homocysteine and Metabolites, and the C-Reactive Protein. Kidney International. 2004;66(1):338-47.

19. Zoccali C, Mallamaci F, Tripepi G. Traditional and Emerging Cardiovascular Risk Factors in End-Stage Renal Disease. Kidney International. 2003;63:S105-S10.

20. Jagieła J, Bartnicki P, Rysz J. Selected Cardiovascular Risk Factors in Early Stages of Chronic Kidney Disease. International Urology and Nephrology. 2020;52(2):303-14.

21. Isbel NM, Haluska B, Johnson DW, Beller E, Hawley C, Marwick TH. Increased Targeting of Cardiovascular Risk Factors in Patients With Chronic Kidney Disease Does Not Improve Atheroma Burden or Cardiovascular Function. American Heart Journal. 2006;151(3):745-53.

22. Weiner DE, Tighiouart H, Stark PC, Amin MG, MacLeod B, Griffith JL, et al. Kidney Disease Is a Risk Factor for Recurrent Cardiovascular Disease and Mortality. American Journal of Kidney Diseases. 2004;44(2):198-206.

23. Bronas UG. Exercise Training and Reduction of Cardiovascular Disease Risk Factors in Patients With Chronic Kidney Disease. Advances in Chronic Kidney Disease. 2009;16(6):449-58.

24. Burnier M, Damianaki A. Hypertension as a Cardiovascular Risk Factor in Chronic Kidney Disease. Circulation Research. 2023;132(8):1050-63.

25. Matsushita K, Ballew SH, Wang AY, Kalyesubula R, Schaeffner E, Agarwal R. Epidemiology and Risk of Cardiovascular Disease in Populations With Chronic Kidney Disease. Nature Reviews Nephrology. 2022;18(11):696-707.

26. Zoccali C, Mark PB, Sarafidis P, Agarwal R, Adamczak M, Bueno de Oliveira R, et al. Diagnosis of Cardiovascular Disease in Patients With Chronic Kidney Disease. Nature Reviews Nephrology. 2023;19(11):733-46.

27. Coresh J, Astor B, Sarnak MJ. Evidence for Increased Cardiovascular Disease Risk in Patients With Chronic Kidney Disease. Current Opinion in Nephrology and Hypertension. 2004;13(1):73-81.

28. De Lima JJ, Gowdak LHW, David-Neto E, Bortolotto LA. Diabetes, Cardiovascular Disease, and Cardiovascular Risk in Patients With Chronic Kidney Disease. High Blood Pressure & Cardiovascular Prevention. 2021;28:159-65.

29. Ocak G, Rookmaaker M, Algra A, De Borst G, Doevendans P, Kappelle L, et al. Chronic Kidney Disease and Bleeding Risk in Patients at High Cardiovascular Risk: A Cohort Study. Journal of Thrombosis and Haemostasis. 2018;16(1):65-73.

30. Carmena R, Ascaso JF, Redon J. Chronic Kidney Disease as a Cardiovascular Risk Factor. Journal of Hypertension. 2020;38(11):2110-21.

31. Luft FC. Renal Disease Is a Risk Factor for Cardiovascular Disease. Basic Research in Cardiology. 2000;95:I72-I6.

32. Wheeler DC, Townend JN, Landray MJ. Cardiovascular Risk Factors in Predialysis Patients: Baseline Data From the Chronic Renal Impairment in Birmingham (CRIB) Study. Kidney International. 2003;63:S201-S3.

33. Subbiah AK, Chhabra YK, Mahajan S. Cardiovascular Disease in Patients With Chronic Kidney Disease: A Neglected Subgroup. Heart Asia. 2016;8(2):56-61.