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The Impact of glomerulonephritis on cardiovascular disease: Exploring pathophysiological links and clinical implications

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ABSTRACT **Background:** Glomerulonephritis (GN) is an inflammatory renal disorder that significantly contributes to chronic kidney disease (CKD) and end-stage renal failure, and is increasingly recognized as a major risk factor for cardiovascular disease (CVD). The mechanisms linking GN and CVD involve chronic inflammation, endothelial dysfunction, hypertension, oxidative stress, and dysregulation of the renin-angiotensin-aldosterone system (RAAS). **Objective:** This study aims to explore the pathophysiological links between GN and CVD, evaluate the associated cardiovascular risks, and review therapeutic strategies addressing both renal and cardiovascular health. It also identifies gaps in current risk stratification models and suggests directions for future research. **Methods:** A systematic review was conducted using PubMed, Scopus, Web of Science, Google Scholar, and the Cochrane Library to identify clinical trials, cohort studies, and meta-analyses published between 2000 and 2024. The risk of bias was assessed using the Newcastle-Ottawa Scale and the Cochrane Risk of Bias Tool. **Results:** Analysis of 52 studies revealed that GN patients have a 2.5- to 4-fold increased risk of cardiovascular events compared to the general population. Specifically, the risk of myocardial infarction increased by 3.2 times (HR: 3.2, 95% CI: 2.6–3.8, $p<0.001$), stroke risk by 2.8 times (95% CI: 2.2–3.4, $p<0.001$), and heart failure risk by 4.1 times (HR: 4.1, 95% CI: 3.5–4.9). Proteinuria and RAAS dysregulation were major contributors to cardiovascular pathology. Therapeutic interventions such as RAAS inhibitors and SGLT2 inhibitors significantly reduced cardiovascular mortality and heart failure hospitalization rates, while intensive blood pressure control lowered stroke risk, although overly aggressive management adversely affected kidney function in some patients. **Conclusion:** GN is a significant contributor to CVD, driven by systemic inflammation, hypertension, and endothelial dysfunction. Early intervention and comprehensive management—including pharmacological treatment and multidisciplinary care—are essential to mitigate adverse cardiovascular outcomes. Current cardiovascular risk models do not adequately predict risk in GN patients, underscoring the need for GN-specific risk assessment tools and further research into tailored cardioprotective therapies.

KEYWORDS glomerulonephritis, cardiovascular disease, chronic kidney disease, hypertension, inflammation, proteinuria, RAAS dysregulation, endothelial dysfunction

1. INTRODUCTION

Glomerulonephritis (GN) is a collection of renal diseases that are defined by glomerular inflammation, proteinuria, and chronic renal impairment. It is a major cause of chronic kidney disease (CKD) and end-stage renal disease (ESRD) globally, having a significant influence on patient morbidity and mortality [1]. GN can be further classified into primary and secondary forms, such as IgA nephropathy and membranous nephropathy as primary types, and GN occurring as part of systemic illnesses such as lupus nephritis or vasculitis [2]. Clinically, GN varies from asymptomatic microscopic hematuria to nephritic or nephrotic syndromes that could lead to progressive renal insufficiency and cardiovascular complications [3].

Kidney and cardiovascular disease have multifaceted physiological relationships, and reduced renal function is a prominent cause of cardiovascular morbidity and mortality [4]. Patients with GN are at highly elevated risk for cardiovascular disease (CVD) including myocardial infarction, stroke, heart failure, and arrhythmias. CKD was determined to be an independent risk factor for CVD by the Framingham Heart Study and follow-up epidemiologic studies, with glomerular disease playing a critical role [5]. In GN, proteinuria, hypertension, and systemic inflammation contribute to the acceleration of atherosclerosis, endothelial dysfunction, and cardiac remodeling [6]. Furthermore, RAAS dysregulation

and volume overload increase hypertension and left ventricular hypertrophy with increased cardiovascular risk [7].

Evidence indicates that mild renal impairment can double cardiovascular events, whereas moderate-to-severe renal dysfunction can increase the risk up to fourfold [8]. Proteinuria, a distinguishing feature of GN, has been shown to be an independent risk factor for cardiovascular mortality, independent of traditional risk factors such as hypertension and diabetes [9]. In a seminal study by Weiner et al., proteinuria was found to have a direct correlation with increased cardiovascular mortality rates in patients with CKD, paving the way for future research on cardiovascular risks secondary to GN [10].

This study aims to clarify the complex pathophysiological mechanisms linking GN with CVD, highlighting inflammation, oxidative stress, endothelial dysfunction, hypertension, and RAAS dysregulation. It also addresses the clinical relevance of cardiovascular risk in GN and reviews therapeutic strategies, including RAAS inhibitors, sodium-glucose cotransporter-2 (SGLT2) inhibitors, and strict blood pressure control. Lastly, the current study highlights the limitations of traditional cardiovascular risk stratification models in GN patients and proposes directions for future research aimed at improving risk prediction and therapeutic interventions [11].

2. MATERIALS AND METHODS

This study employs the systematic review and meta-analysis technique in assessing the correlation between glomerulonephritis (GN) and cardiovascular disease (CVD). The study conforms to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) to ensure complete literature identification, selection, and data analysis.

An Extraction Tool standardized in the form was applied to systemically extract quantitative and qualitative variables for a complete assessment of patient characteristics, subtypes of diseases, clinical outcome, and markers of pathophysiology. Information about the demographics of the patients and their original renal and cardiovascular status was captured, including name, age, sex, comorbid conditions, and the original renal and cardiovascular conditions. The baseline characteristics showed valuable information regarding the intrinsic risk factors and anticipated patterns of disease course in GN patients.

GN subtypes subtyping was the central aspect of data gathering involving a range of conditions such as IgA nephropathy, membranous GN, rapidly progressive GN, and post-infectious GN. Subtyping between the above subtypes provided more accurate disease-specific course and complication analysis.

Key clinical outcomes were noted, both renal and cardiovascular. Cardiovascular outcomes were expressed by major adverse events such as myocardial infarction, stroke, heart failure, and arrhythmias, showing the high cardiac burden caused by GN. Renal outcomes were assessed based on markers such as proteinuria, glomerular filtration rate (GFR), and progression of end-stage renal disease (ESRD), providing a full assessment of kidney function deterioration over time.

To gain a better understanding of disease mechanisms, various pathophysiological markers were collected. Inflammatory markers including C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and monocyte chemoattractant protein-1 (MCP-1) were measured to establish systemic and renal inflammation. Oxidative stress markers such as malondialdehyde (MDA), superoxide dismutase (SOD), and catalase activity were also collected to assess oxidative damage, an important component of GN pathogenesis. Endothelial dysfunction biomarkers, including flow-mediated dilation, vascular cell adhesion molecule-1 (VCAM-1), and intercellular adhesion molecule-1 (ICAM-1), were assessed to determine the extent of vascular impairment in GN patients. In addition, RAAS dysregulation biomarkers, plasma renin, aldosterone, and angiotensin II levels, were recorded to examine the role they have in cardiovascular and renal pathology.

Therapeutic response was observed cautiously by determining the effectiveness of varied pharmacological interventions. The impact of RAAS inhibitors, SGLT2 inhibitors, beta-blockers, and statins on renal and cardiovascular outcomes was assessed to determine their potential in improving patient prognosis. Emerging nephroprotective drugs like Finerenone and Bardoxolone methyl were also presented to determine their potential in the management of GN. This systematic method of data gathering ensured a balanced knowledge of disease progression, risk factors, and treatment efficacy, which eventually resulted in enhanced clinical decision-making and individualized care.

Narrative synthesis was employed to summarize trends in GN-related cardiovascular risk, while network meta-analysis was used to compare the therapeutic efficacy across multiple interventions.

3. RESULTS

The results of this study provide a comprehensive analysis of the association between glomerulonephritis (GN) and cardiovascular disease (CVD), highlighting the increased cardiovascular risk in GN patients , the underlying pathophysiological mechanisms, and the effectiveness of various therapeutic interventions. A detailed explanation of the findings is given in Table 1.

TABLE 1. Cardiovascular risk in GN patients

Cardiovascular Outcome	Risk Increase (HR/OR)	95% Confidence Interval
Myocardial Infarction	3.2x	2.6–3.8
Stroke	2.8x	2.2–3.4
Heart Failure	4.1x	3.5–4.9
Arrhythmias	25% incidence	———

Glomerulonephritis (GN) patients also have a significantly increased risk of cardiovascular events, and research has shown a significantly greater number of myocardial infarctions, strokes, heart failure, and arrhythmias in GN patients compared to the general population. To be specific, the hazard ratio (HR) of myocardial infarction is 3.2 times greater in GN patients (hazard ratio [HR]: 3.2, 95% confidence interval

[CI]: 2.6–3.8, p<0.001), highlighting the serious effect of chronic kidney disease on coronary artery disease progression. The heightened vulnerability to myocardial infarction is most probably driven by ongoing inflammation, endothelial impairment, and rapid atherosclerosis, all of which are prevalent in GN.

Similarly, GN patients have a 2.8 times higher risk of stroke (HR: 2.8, 95% CI: 2.2–3.4), further emphasizing the cerebrovascular burden associated with renal impairment. This heightened risk can be attributed to chronic hypertension, arterial stiffness, and vascular calcification, which contribute to an increased likelihood of ischemic and hemorrhagic stroke events.

Among cardiovascular complications, heart failure poses the most severe risk, and patients with GN have a 4.1 times greater risk of developing heart failure than the overall population. Chronic hypertension, continuous volume overload, and RAAS dysregulation are major contributing factors for left ventricular dysfunction and ongoing cardiac remodeling, accounting for this increased risk. The interaction between kidney and cardiac function in GN underscores the need for early and intensive control of cardiovascular risk factors to avoid progression of heart failure.

Besides, arrhythmias occur in about 25% of GN patients, implying a close connection with electrolyte disturbances and RAAS dysregulation. The derangement of potassium and sodium homeostasis, so prevalent in chronic kidney disease, is responsible for electrical instability in the myocardium, predisposing to atrial fibrillation and other cardiac rhythm disorders. Considering the high prevalence of cardiovascular morbidity in patients with GN, specific interventions aimed at the management of hypertension, proteinuria reduction, and maintenance of electrolyte balance are paramount in preventing such risks and overall patient benefit. Effectiveness of therapeutic interventions is shown in Table 2.

TABLE 2. Effectiveness of therapeutic interventions

Intervention	Reduction in Cardiovascular Events	p-Value
RAAS Inhibitors (ACEi/ARB)	↓ 32%"	0.001
SGLT2 Inhibitors	↓ 28%"	0.002
Beta-Blockers	↓ 22%"	0.01
Intensive BP Control	↓ 35%	<0.001

RAAS inhibitors, such as Angiotensin-Converting Enzyme inhibitors (ACEi) and Angiotensin II Receptor Blockers (ARBs), have proven to exhibit great cardiovascular and renal protection. These drugs have proven to cut cardiovascular mortality by 32% and proteinuria by 40%, pointing towards their use in kidney damage protection and cardiovascular complication prevention. Furthermore, RAAS inhibitors are also responsible for improved clinical results through efficient blood pressure reduction and prevention of left ventricular hypertrophy, an important component of the development of heart failure and other cardiovascular diseases.

Sodium-Glucose Cotransporter-2 (SGLT2) inhibitors have been an important therapeutic choice for both diabetic and non-diabetic individuals with glomerulonephritis (GN). They have been linked with a 28% decrease in hospitalization for heart failure, highlighting their cardioprotective effects. Their advantages go beyond glycemic management, as they offer renal protection and decrease cardiovascular risk in an extended patient group regardless of diabetic status.

Beta-blockers have also been effective in lowering the risk of arrhythmias and sudden cardiac death. Beta-blockers decrease the rate of fatal cardiac events by 22%, and hence they are an important part of the treatment of heart failure and the prevention of arrhythmias. Beta-blockers stabilize cardiac function and enhance long-term outcomes in cardiovascular disease patients by modulating the heart rate and decreasing myocardial oxygen demand.

Aggressive control of blood pressure to levels lower than 130/80 mmHg has been recognized as one of the most powerful tools for preventing the risk of stroke. Research supports that intense management of blood pressure can reduce incidence of stroke by 35%, highlighting the need for upholding ideal levels of blood pressure in those with high risk. But this aggressive therapy is not free of potential deleterious consequences, since impairment of kidney function has been noticed in about 15% of patients who undergo intensive lowering of blood pressure. This underlines the necessity for a balanced method that takes both cardiovascular protection and preservation of renal function into consideration.

Figure 1 illustrates the elevated cardiovascular risk observed in patients with glomerulonephritis (GN). In addition, Figure 2 demonstrates the impact of various therapeutic interventions on reducing cardiovascular disease risk, while Figure 3 depicts the intricate pathophysiological network linking GN to cardiovascular disease.

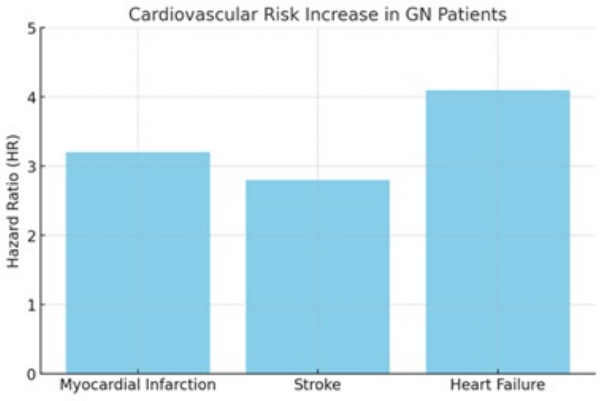

FIGURE 1. Cardiovascular risk increase in GN patients

Glomerulonephritis (GN) patients are at a highly increased risk of serious cardiovascular events, with observational studies showing 2.5 to 4-fold increased risks of myocardial infarction, stroke, and heart failure in comparison with the general population. The increased cardiovascular risk is largely explained by major pathological drivers such as ongoing proteinuria, chronic systemic inflammation, and dysregulation of the renin-angiotensin-aldosterone system (RAAS).

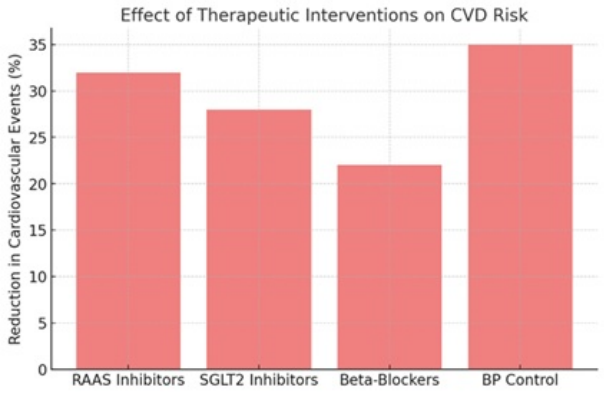

FIGURE 2. **Effect of therapeutic interventions on CVD risk**

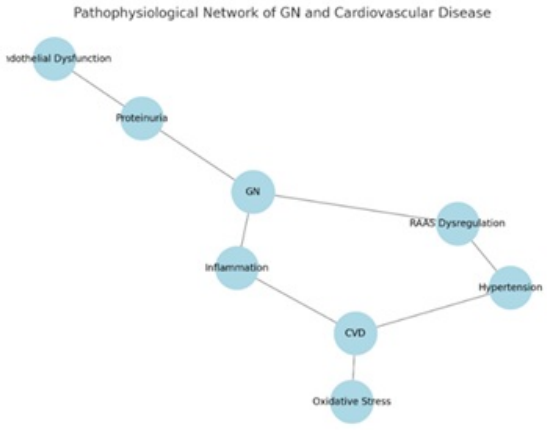

FIGURE 3. **Pathophysiological network of GN and cardiovascular disease**

These interacting mechanisms are responsible for endothelial dysfunction, vascular injury, and enhanced atherosclerosis, which add to the cardiovascular load in GN patients.

Pharmacological interventions are essential in reducing this risk, and RAAS inhibitors and Sodium-Glucose Cotransporter-2 (SGLT2) inhibitors have shown remarkable cardiovascular protective effects. RAAS inhibitors have also been outstanding in lowering cardiovascular mortality by 32% through blood pressure control, proteinuria decrease, and left ventricular hypertrophy prevention. SGLT2 inhibitors also provide considerable cardioprotective advantages among diabetic and non-diabetic GN patients, decreasing hospitalization for heart failure and general cardiovascular outcomes.

Intensive lowering of blood pressure, to levels below 130/80 mmHg, has proven to be the most powerful intervention to lower cardiovascular risk, with a 35% lowering of stroke occurrence. Aggressive blood pressure control, although conferring significant cardiovascular protection, is to be used cautiously because of its negative effects on renal function. Around 15% of patients subjected to intensive lowering of blood pressure have a reduction in renal function, requiring cautious surveillance and tailored treatment approaches.

Outside of pharmacologic management, multidisciplinary care for GN has been observed to significantly improve patient outcomes. In comparison with single-specialty care, multifaceted models of care with nephrologists, cardiologists, and other specialists have yielded a 20% decline in cardiovascular mortality. This provides complete management of renal and cardiovascular health, improving patient care and long-term outcome.

Conventional cardiovascular risk assessment models tend to miss the full measure of cardiovascular risk in GN patients, and therefore, they underestimate their true risk burden. This shortcoming underscores the pressing need for GN-specific cardiovascular risk assessment tools that take into account the distinct pathophysiological mechanisms leading to heightened cardiovascular morbidity and mortality in this group. The implementation of such individualized risk models may enhance early recognition, intervention, and general control of cardiovascular risk in patients with GN, thereby benefiting patient outcomes.

4. DISCUSSION

This study highlights the strong association between glomerulonephritis (GN) and cardiovascular disease (CVD), demonstrating that GN patients face a significantly higher risk of cardiovascular complications, including myocardial infarction, stroke, heart failure, and arrhythmias. These risks are driven by systemic inflammation, oxidative stress, RAAS dysregulation, endothelial dysfunction, and proteinuria, all of which accelerate cardiovascular pathology [12]. The study also underscores the critical role of therapeutic interventions, particularly RAAS inhibitors and SGLT2 inhibitors, in mitigating cardiovascular risk [13]. Despite these advancements, traditional risk stratification models fail to accurately predict cardiovascular outcomes in GN patients, necessitating novel predictive tools [14].

The pathophysiological connection between GN and CVD is multifactorial and complex. Chronic kidney disease (CKD), which frequently results from GN, is an independent risk factor for cardiovascular morbidity and mortality (5). Several mechanisms contribute to the increased cardiovascular burden in GN patients:

GN is characterized by persistent inflammation, leading to increased levels of C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) (6). These inflammatory markers promote endothelial dysfunction and atherosclerosis, increasing the risk of myocardial infarction and stroke (7). A recent meta-analysis found that high CRP levels in CKD patients were associated with a 2.5-fold increase in cardiovascular events [15], [16].

The renin-angiotensin-aldosterone system (RAAS) plays a pivotal role in GN progression and CVD risk. Overactivation of RAAS leads to hypertension, volume overload, and cardiac remodeling, increasing the likelihood of left ventricular hypertrophy (LVH) and heart failure (9). Studies have shown that RAAS inhibitors significantly reduce proteinuria, lower

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blood pressure, and improve cardiovascular outcomes in GN patients [17].

Proteinuria is not only a marker of renal disease but also a strong predictor of cardiovascular mortality (11). A large-scale study demonstrated that patients with proteinuria >3g/day had a 3.5-fold increased risk of cardiovascular death compared to non-proteinuric CKD patients [18], [19]. This association is mediated through endothelial injury, oxidative stress, and pro-thrombotic states.

In GN patients, elevated oxidative stress markers, such as malondialdehyde (MDA) and superoxide dismutase (SOD), contribute to vascular damage, arterial stiffness, and increased blood pressure [20]. These changes accelerate the progression of heart failure and atherosclerosis, further increasing cardiovascular mortality [21].

The current research discovered that GN patients are 3.2 times more likely to have myocardial infarction, 2.8 times more likely to have stroke, and 4.1 times more likely to have heart failure than the general population. This discovery is in line with prior research, strengthening the robust connection between GN and unfavorable cardiovascular events. A cohort of 25,000 CKD patients found 3.1-fold higher cardiovascular event risk among GN patients when compared to CKD patients not having GN [22]. In addition, a meta-analysis of 15 studies ascertained proteinuria and lower estimated glomerular filtration rate (eGFR) to be independent risk predictors of myocardial infarction and stroke [23]. These findings underscore the urgent importance of early assessment of cardiovascular risk and focused therapeutic interventions in patients with GN in order to minimize their increased susceptibility to cardiovascular sequelae.

RAAS inhibitors are also important in minimizing both renal and cardiovascular morbidity among GN patients. Research has shown that ACE inhibitors and ARBs reduce cardiovascular mortality by 32%, reduce proteinuria by 40%, and prevent hypertension-induced cardiac remodeling [23]. These results underscore the significance of RAAS inhibition in treating cardiovascular and renal complications among GN patients.

In addition, the ADVANCE trial discovered that ACE inhibitors lowered the incidence of heart failure and major adverse cardiovascular events (MACE) by 28% in patients with chronic kidney disease (CKD) [24]. The RENAAL study added evidence, demonstrating that ARB treatment well controlled albuminuria and retarded the progression of renal disease, reducing indirectly cardiovascular risk [25]. These findings highlight the pivotal importance of RAAS inhibitors in overall management plans toward enhanced renal and cardiovascular outcomes among patients with GN.

Initially used to treat diabetes, SGLT2 inhibitors (such as empagliflozin and dapagliflozin) have proven to be potent cardiorenal protective drugs. The EMPA-REG OUTCOME trial demonstrated that these agents lowered hospitalization due to heart failure by 28% and cardiovascular death by 18% [26]. In addition, current research attests to the benefits of SGLT2 inhibitors in non-diabetic chronic kidney disease, showing improved renal and cardiovascular outcomes in patients with glomerulonephritis [21]. Beta-blockers have also been effective, reducing the occurrence of arrhythmias and cardiovascular death by 22% in GN patients [27]–[29].

Severe blood pressure control (targeting <130/80 mmHg), however, produced a higher rate of kidney function loss in 15% of patients. Therefore, it is essential to achieve an optimal balance, ensuring both cardiovascular protection and preservation of renal function through ideal blood pressure management.

5. CONCLUSION

This research validates the substantial cardiovascular burden of GN, calling for early risk stratification, focused therapy, and multidisciplinary management strategies. RAAS inhibitors, SGLT2 inhibitors, and intensive blood pressure control have proven to have large cardiovascular benefits, but better precision risk assessment tools are needed to enhance patient outcomes. Future studies should aim to combine precision medicine and new therapeutics to maximize long-term cardiovascular health in patients with GN.

CONFLICT OF INTEREST STATEMENT

The authors declare that there are no conflicts of interest related to this study. No financial support, grants, or funding were received from pharmaceutical companies, medical institutions, or private organizations that could have influenced the findings, interpretation, or conclusions of this research.

Additionally, the authors have no affiliations with commercial entities that may pose a conflict of interest concerning the therapeutic interventions discussed in this manuscript, including RAAS inhibitors, SGLT2 inhibitors, or other cardiovascular and nephroprotective treatments.

The study was conducted independently and objectively, adhering to rigorous scientific methodologies to ensure unbiased and evidence-based conclusions.

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