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ORIGINAL RESEARCH

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Relationship between proteinuria, physical activity, and left ventricular hypertrophy in patients with chronic glomerulonephritis

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Abstract

Background: Chronic glomerulonephritis (GN) is a kidney disorder characterized by glomerular inflammation and damage, often triggered by immune-mediated mechanisms and infections. The relationship between proteinuria, physical activity, and left ventricular hypertrophy (LVH) remains unclear. This study aimed to investigate the association between proteinuria and LVH in non-diabetic patients with chronic GN and to assess the impact of physical activity on cardiovascular health. **Methods**: A total of 165 male chronic GN patients (mean age: 55.65 $\pm$ 15.81 years) were compared with 90 age-matched healthy males. Proteinuria was measured using 24-hour quantification (24 h QP). Echocardiographic parameters, including left ventricular ejection fraction (LVEF), left ventricular mass index (LVMI), and LVH, were assessed. Physical activity levels were evaluated using a validated self-reported questionnaire. **Results**: Proteinuria $\geq$3.5 g/day (nephrotic range) was observed in 54 (32.7%) patients. Chronic GN patients had significantly higher creatinine, LVMI, and LVH compared to controls. Patients with nephrotic proteinuria exhibited greater LVMI and LVH than those with non-nephrotic proteinuria. A significant association was observed between LVMI and LVH in patients with nephrotic syndrome. Univariate regression analysis showed that increased LVMI and LVH were associated with higher nephrotic proteinuria. Notably, higher physical activity was associated with reduced nephrotic proteinuria and lower for cardiovascular health. **Conclusions**: LVH was more prevalent among patients with chronic GN, particularly those with nephrotic proteinuria. The findings underscore the importance of physical activity in reducing cardiovascular risks in this population. These results support the integration of tailored exercise programs into the clinical management of chronic kidney disease (CKD) patients.

Keywords

Chronic glomerulonephritis; Nephrotic proteinuria; Left ventricular mass index; Left ventricular hypertrophy; Physical activity

1. Introduction

Chronic glomerulonephritis (GN) is a rare kidney disease associated with significant morbidity and mortality. GN represents a heterogeneous group of diseases characterized by varying degrees of hematuria, proteinuria, hypertension, and declining renal function. It is the most common cause of end-stage renal failure after diabetes and hypertension. Membranous nephropathy, focal segmental glomerulosclerosis (FSGS), and IgA nephropathy (IgAN) are among the primary causes of GN. Renal biopsy remains the gold standard diagnostic method, aiding in determining specific diagnoses and assessing disease activity and severity [1]. Cardiovascular (CV) disorders are frequently observed in chronic GN patients, including hypertension, cardiac hypertrophy, electrocardiographic abnormalities, and heart failure. Increased urinary albumin excretion is considered a critical indicator of renal dysfunction in these patients. Proteinuria is a significant predictor of coronary heart disease, all-cause mortality, and CV disease [2].

Echocardiography is a preferred imaging modality for cardiac disease due to its low cost, non-invasive nature, safety, and repeatability. Although advanced techniques such as cardiac magnetic resonance imaging (MRI) and computed tomography offer higher resolution and tissue characterization, their high cost, radiation exposure, and patient reluctance limit their clinical application [3]. Thus, echocardiography continues to be widely used for CV screening, particularly in developing countries. In patients with chronic kidney disease (CKD) and those undergoing dialysis, echocardiographic parameters such as left atrial diameter (LAD), left ventricular (LV) mass index

(LVMI), and LV systolic function are reliable indicators for cardiac assessment [4].

Left ventricular hypertrophy (LVH) is more prevalent in CKD patients than in the general population, affecting approximately 40–78% of patients. As CKD progresses, LVMI gradually increases. LVH is a marker of subclinical organ damage associated with hypertension, and these patients face an increased risk of ischemic heart disease, cerebrovascular events, and heart failure. Additionally, LVH is a significant determinant of CV mortality and morbidity in the general population, in non-diabetic hypertensive individuals, and in diabetic patients with microalbuminuria or macroalbuminuria [5].

The role of physical activity in modulating the relationship between proteinuria and LVH has not been well explored. While physical activity has known positive effects on the CV system [6–11], it is unclear how pathological changes, such as those induced by proteinuria, might influence or alter the adaptive physiological responses observed in physically active individuals.

It is known that the incidence of CV diseases, especially coronary artery disease and acute coronary syndromes, is lower in women than in men [12]. The literature indicates that men have a higher risk of developing proteinuria and LVH compared to women [13]. The protective effect of estrogen in premenopausal women plays an important role in cardiovascular outcomes [14]. In our study, we included only male patients in order to eliminate these gender differences.

This study aims to investigate the relationship between proteinuria, physical activity, and left ventricular hypertrophy in male patients with chronic glomerulonephritis.

2. Materials and methods

2.1 Research groups

This study was conducted on a total of 165 male patients who were followed up in the Nephrology Outpatient Clinic due to chronic glomerulonephritis (GN) between March 2022 and November 2023. The patient group was selected from patients who underwent a kidney biopsy and were diagnosed with chronic glomerulonephritis. The patients were compared with 90 healthy male individuals without a history of known comorbidities or medication use. Male patients included in the study were aged over 18 years, had an estimated glomerular filtration rate (eGFR) $\geq$60 mL/min/1.73 m^2, and had no previous history of pacemaker implantation, coronary artery disease, or cardiac interventions. Participants were excluded if they had active infection or malignancy within the last 3 months, cerebrovascular disease, severe ischemic heart disease, diabetic cardiomyopathy, hypertrophic cardiomyopathy, congenital heart disease, significant valvular disease, severe arrhythmia, or left ventricular (LV) ejection fraction (LVEF) $<$50% as determined by echocardiography. Written informed consent was obtained from all participants after the study had been explained to them. Ethics approval (No. E2-22-1196) was received for the study from the ethics committee of Ankara Bilkent City Hospital.

2.2 Physical activity assessment

When the patients first presented to receive their kidney biopsy results, a physical activity survey was performed before the initiation of treatment. The physical activity levels of the patients were evaluated using a validated self-reported physical activity questionnaire. The levels of physical activity were categorized as follows:

Low activity: $<$150 minutes of moderate-intensity physical activity per week.

Moderate activity: 150–300 minutes of moderate-intensity physical activity per week.

High activity: $>$300 minutes of moderate or high-intensity physical activity per week [15].

2.3 Clinical and laboratory measurements

Blood samples were collected from all participants after an 8-hour fast and stored at −80 degrees Celsius for further analysis. Serum creatinine levels were measured using standard laboratory methods. Proteinuria levels were measured using 24-hour proteinuria quantification (24 h QP). For this measurement, urine was collected over 24-hour period. After discarding the first urine void, subsequent urine samples were collected over the next 24 hours, and the samples were sent for analysis after adding the final urine void to the 24-hour urine collection. The 24-hour urine samples from all patients were collected in the hospital and evaluated on the same day as collection. Urinary protein levels were measured using the colorimetric method on the Advia 2400 autoanalyzer (Siemens Diagnostics, Tarrytown, NY, USA). Patients were divided into two groups based on their proteinuria values. Those with proteinuria levels $\geq$3.5 g/day were classified as nephrotic-range proteinuria, while those with proteinuria levels $<$3.5 g/day were classified as non-nephrotic proteinuria. The eGFR value was determined according to previously established protocols [16].

2.4 Echocardiography

All participants underwent transthoracic echocardiographic examination using a commercially available ultrasound system (Sequoia; Siemens Medical Solutions, Mountain View, CA, USA). All measurements were performed in accordance with the recommendations of the American Society of Echocardiography (ASE) [3] and were independently evaluated by two experienced cardiologists who were blinded to the clinical data.

Left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVEDD), interventricular septal thickness (IVST), and posterior wall thickness (PWT) were assessed using standard two-dimensional and Doppler echocardiographic techniques.

Left ventricular mass (LVM) was calculated using the Devereux-modified ASE formula (Eqn. 1) [17]:

$$LVM(g) = 0.8 \times \{1.04 \times [(LVEDD + IVST + PWT)^3 - (LVEDD)^3]\} + 0.6 \quad (1)$$

Left ventricular mass index (LVMI) was obtained by divid-

ing LVM by body surface area (BSA) (Eqn. 2):

$$LVMI\ (g/m^2) = LVM \div BSA \quad (2)$$

Left ventricular hypertrophy (LVH) was defined as an LVMI >115 g/m^2 in men.

2.5 Statistical analysis

Data were analyzed using SPSS software (version 24; IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was used to evaluate the normality of the data distribution. The relationships between categorical variables were assessed using Pearson's chi-square test. Comparisons between groups were performed using the Mann-Whitney U test and the Kruskal-Wallis H test. A p-value < 0.05 was considered statistically significant.

3. Results

The study included a total of 165 male chronic GN patients, with a mean age of 55.65 $\pm$ 15.81 years. The patients were compared with a control group of 90 healthy male individuals matched for age. Of the patients, 27 (16.4%) were using angiotensin-converting enzyme inhibitor (ACE inhibitors), angiotensin II receptor blocker (ARBs) were 21 (12.7%), were using calcium-channel blockers 17 (10.3%), 5 (3%) were using alpha-blockers, 4 (2.4%) were using beta-blockers and 5 (3%) were using diuretics. Regarding the etiologies of chronic GN, 43 (26.1%) had focal segmental glomerulosclerosis (FSGS), 19 (11.5%) had hypertensive nephropathy, 8 (4.8%) had amyloidosis, 7 (4.2%) had systemic lupus erythematosus (SLE) nephritis, 21 (12.7%) had IgA nephropathy (IgAN), 27 (16.4%) had membranous nephropathy, 15 (9.1%) had membranoproliferative nephropathy (MPGN), 6 (3.6%) had minimal change nephropathy, 11 (6.7%) had crescentic glomerulonephritis and 8 (4.8%) had tubulointerstitial nephritis. In 54 (32.7%) of the patients, proteinuria was determined to be $\geq$3.5 g/day. Compared with healthy individuals, patients had significantly higher creatinine ($p < 0.001$), LVMI ($p = 0.026$), and LVH ($p = 0.002$), while eGFR ($p < 0.001$) was significantly lower. No significant differences were found between the two groups regarding LVEF, LVEDD, and LVESD (Left Ventricular End-Systolic Diameter) ($p > 0.05$) (Table 1).

3.1 Nephrotic vs non-nephrotic proteinuria

In the comparison between patients with nephrotic and non-nephrotic proteinuria, LVMI ($p = 0.038$) and LVH ($p = 0.003$) were significantly higher in patients with nephrotic proteinuria compared with those with non-nephrotic proteinuria. No significant differences were observed between the two groups regarding age and eGFR ($p > 0.05$) (Table 2).

3.2 Logistic regression analysis

According to the results of the univariate logistic regression analysis, as the LVH value increased, there was a 1.009-fold increase in the likelihood of nephrotic proteinuria (Odds ratio (OR) = 1.009, 95% Confidence interval (CI) 1.003–1.020). Similarly, as the LVMI increased, there was a 1.002-fold increase in nephrotic proteinuria (OR = 1.002, 95% CI 0.981–1.020). However, in the multivariate analysis, the effect of LVMI and LVH on the level of proteinuria was not statistically significant (Table 3).

3.3 Physical activity analysis

According to the analysis based on physical activity levels, patients were divided into low, medium, and high activity groups, with 55 patients in each group. Among the patients with low physical activity, the rate of nephrotic proteinuria was 50%, the mean left ventricular mass index (LVMI) was 140.5 g/m^2, and the rate of left ventricular hypertrophy (LVH) was 65%. With moderate physical activity, these values decreased to 35%, 130.2 g/m^2, and 50%, respectively, while with high physical activity, the nephrotic proteinuria rate was 15%, the mean LVMI was 120.8 g/m^2, and the LVH rate was 30%. These results show that an increase in physical activity levels are associated with lower rates of nephrotic proteinuria and LVH, supporting the positive effect of physical activity on cardiovascular health (Table 4).

4. Discussion

In our study, increased LVMI and LVH development were observed in non-diabetic chronic GN male patients with an eGFR value $\geq$60 mL/min/1.73 m^2 compared to healthy male individuals. It was observed that patients with nephrotic proteinuria had higher LVMI values and a greater prevalence of LVH compared to those with non-nephrotic proteinuria. A significant relationship was observed between nephrotic proteinuria and LVMI as well as LVH (Fig. 1). However, no relationship was found between non-nephrotic proteinuria and LVH (Fig. 2).

LVH is an indicator of subclinical organ damage related to hypertension and is an independent predictor of CV morbidity and mortality [5]. It is known that LVH determined by echocardiography, a non-invasive method, can be used to predict CV mortality risk in the general population with various diseases. Therefore, in our study, the cardiac parameters of patients were determined using echocardiography. Schroeder *et al.* [18] reported increased LVMI values in patients with mild-to-moderate renal dysfunction in chronic GN, independent of renal function, compared to healthy individuals. Stefanski *et al.* [19] reported that cardiac remodeling began to develop even in those with normal blood pressure, normal GFR, and non-nephrotic proteinuria in the early stages of IgAN. In a study that followed 436 SLE nephritis patients without a history of cardiac disease for 18 months. They reported that LVH developed in 182 (41.74%) patients, which was higher than that reported in a previous study. The authors suggested that this could be due to higher levels of proteinuria and more advanced renal failure in these patients. They reported that increased LVH was associated with increased cardiac mortality [20]. Sági *et al.* [2] followed a total of 67 patients with IgAN for 184 months. They reported that baseline kidney function was an independent determinant of LVH development. They

TABLE 1. Demographic, clinical, and echocardiographic findings of the patient and control groups.

	Patients (n = 165) Mean ± S.D./n (%)	Healthy control group (n = 90) Mean ± S.D./n (%)	*p*
Age (yr)	55.65 ± 15.81	56.52 ± 14.05	0.223*
Medications			
ACE inh/ARB	27 (16.4%)/21 (12.7%)		
Ca-channel blocker	17 (10.3%)		
Alpha-blocker	5 (3.0%)		
Beta-blocker	4 (2.4%)		
Diuretic	5 (3.0%)		
FSGS	43 (26.1%)		
Hypertensive nephropathy	19 (11.5%)		
Amyloidosis	8 (4.8%)		
SLE nephritis	7 (4.2%)		
IgAN	21 (12.7%)		
Membranous nephropathy	27 (16.4%)		
MPGN	15 (9.1%)		
Minimal change nephropathy	6 (3.6%)		
Crescentic glomerulonephritis	11 (6.7%)		
Tubulointerstitial nephritis	8 (4.8%)		
Creatinine (mg/dL)	1.03 ± 0.28	0.86 ± 0.20	<0.001*
eGFR (mL/min/1.73 m^2)	79.12 ± 23.29	95.91 ± 96.88	<0.001*
Proteinuria			
<3.5 g/day	111 (67.3%)		
≥3.5 g/day	54 (32.7%)		
LVEF (%)	58.38 ± 6.59	59.11 ± 5.05	0.980*
LVEDD (mm)	4.57 ± 0.46	4.57 ± 0.43	0.301*
LVESD (mm)	2.89 ± 0.69	2.99 ± 0.52	0.222*
LVMI (g/m^2)	130.96 ± 41.01	127.73 ± 24.40	0.026*
LVH	184.48 ± 55.72	166.03 ± 50.46	0.002*

**Mann-Whitney U test, Mean ± standard deviation (S.D.). ACE inh: angiotensin-converting enzyme inhibitor; ARB: angiotensin II receptor blocker; FSGS: focal segmental glomerulosclerosis; SLE: systemic lupus erythematosus; IgAN: IgA nephropathy; MPGN: membranoproliferative nephropathy; eGFR: estimated glomerular filtration rate; LVEF: left ventricular ejection fraction; LVEDD: left ventricular end-diastolic dimension; LVMI: left ventricular mass index; LVH: left ventricular hypertrophy; LVESD: Left Ventricular End-Systolic Diameter. *: Statistical significance was defined as $p < 0.05$.*

TABLE 2. Comparison of clinical and echocardiographic findings according to nephrotic and non-nephrotic proteinuria levels.

	Non-nephrotic proteinuria (<3.5 g/day) (n = 111)	Nephrotic proteinuria (≥3.5 g/day) (n = 54)	*p*
Age (yr)	49.97 ± 15.75	52.04 ± 16.00	0.548*
eGFR (mL/min/1.73 m^2)	79.61 ± 23.95	78.11 ± 22.04	0.879*
LVEF (%)	58.76 ± 6.66	57.61 ± 6.46	0.150*
LVEDD (mm)	4.63 ± 0.50	4.65 ± 0.36	0.521*
LVESD (mm)	2.88 ± 0.72	2.93 ± 0.61	0.460*
LVMI (g/m^2)	127.24 ± 19.54	129.64 ± 32.08	0.038*
LVH	175.63 ± 54.38	202.67 ± 54.48	0.003*

**Mann-Whitney U test, Mean ± standard deviation. eGFR: estimated glomerular filtration rate; LVEF: left ventricular ejection fraction; LVEDD: left ventricular end-diastolic dimension; LVMI: left ventricular mass index; LVH: left ventricular hypertrophy; LVESD: Left Ventricular End-Systolic Diameter. *: Statistical significance was defined as $p < 0.05$.*

TABLE 3. Relationship between echocardiography findings and nephrotic proteinuria in multivariate analysis.

	Univariate		Multivariate	
	OR (95% CI)	*p*	OR (95% CI)	*p*
LVMI (g/m^2)	1.002 (0.981−1.020)	0.043	1.006 (0.984−1.030)	0.828
LVH	1.009 (1.003−1.020)	0.005	1.010 (1.000−1.020)	0.604

OR: Odds ratio; CI: Confidence interval; LVMI: left ventricular mass index; LVH: left ventricular hypertrophy. Accuracy = 0.635.

TABLE 4. Grouping according to physical activity levels and analysis of proteinuria and LVH.

Physical activity level	Nephrotic Proteinuria (%)	LVMI (g/m^2, Average)	LVH (%)
Low (n = 55)	50%	140.5	65%
Moderate (n = 55)	35%	130.2	50%
High (n = 55)	15%	120.8	30%

LVMI: left ventricular mass index; LVH: left ventricular hypertrophy.

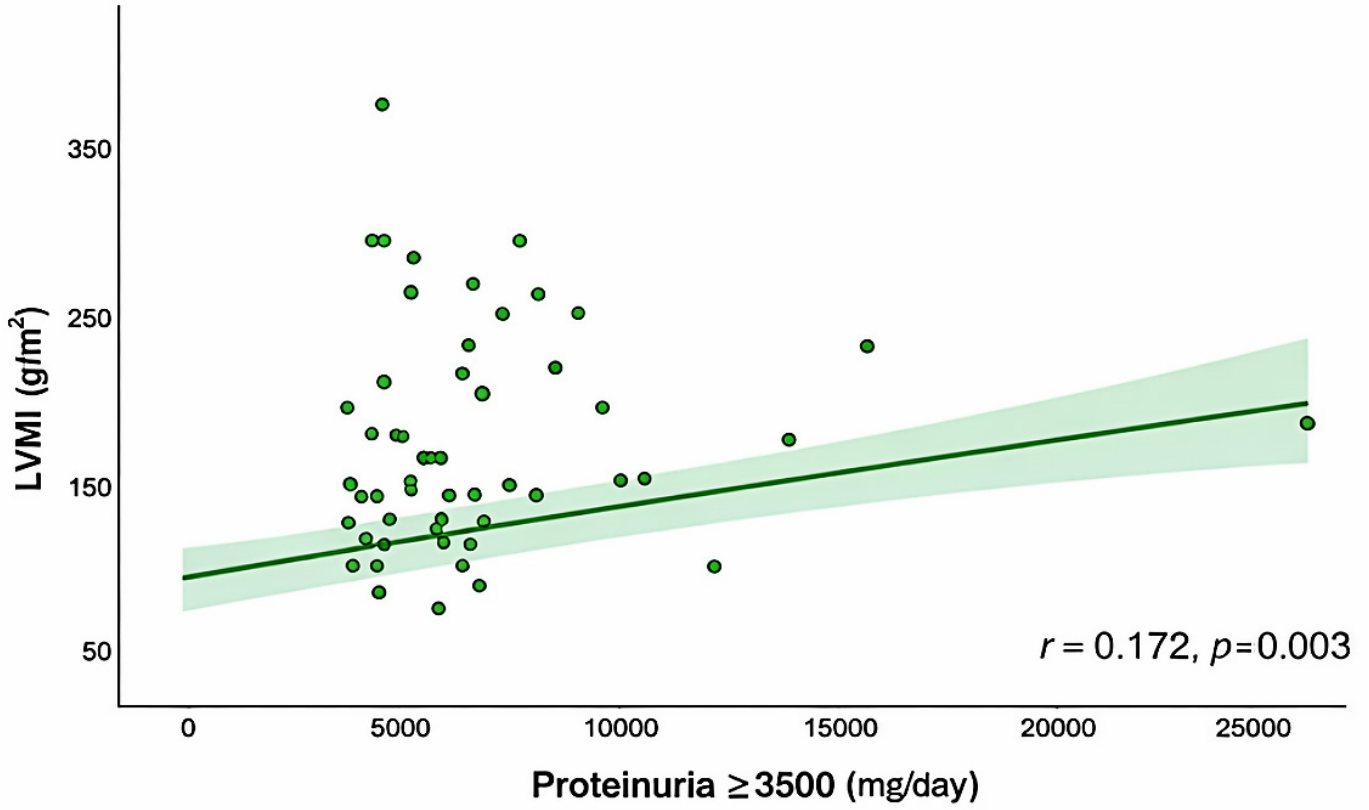

FIGURE 1. Relationship between nephrotic proteinuria and LVH. A significant correlation was found between nephrotic proteinuria and LVMI (r = 0.172, p = 0.003) and LVH (r = 0.167, p = 0.002). LVMI: left ventricular mass index.

also reported that increased LVMI had a significant impact on both combined and renal and CV outcomes in male patients. The authors suggested that the detection of LVH could be a useful parameter not only for CV risk assessment but also for classifying renal risk in the development of renal failure [2]. In our study, similarly, increased LVMI and LVH development were observed in non-diabetic chronic GN patients compared to healthy individuals.

Bhusal *et al*. [21] reported no relationship between microalbuminuria and LVH in non-diabetic hypertensive individuals. Sági *et al*. [2] also reported no relationship between proteinuria and LVMI in a study conducted in patients with IgAN. In contrast to these studies, Al-Sharifi *et al*. [22] followed 100 hypertensive patients for 7.2 years and reported a relationship between microalbuminuria and LVH. In their study on 870 hypertensive patients [22], the authors reported a relationship between both microalbuminuria and macroalbuminuria and LVH. They reported that this relationship was particularly more common in patients with hypertension duration less than 15 years and those under the age of 70 years [23]. Yan *et al*. [24] reported a relationship between microalbuminuria and LVH in women; however, this relationship was not observed in men. Salmasi *et al*. [25] reported that as the degree of albuminuria increased in hypertensive diabetic patients,

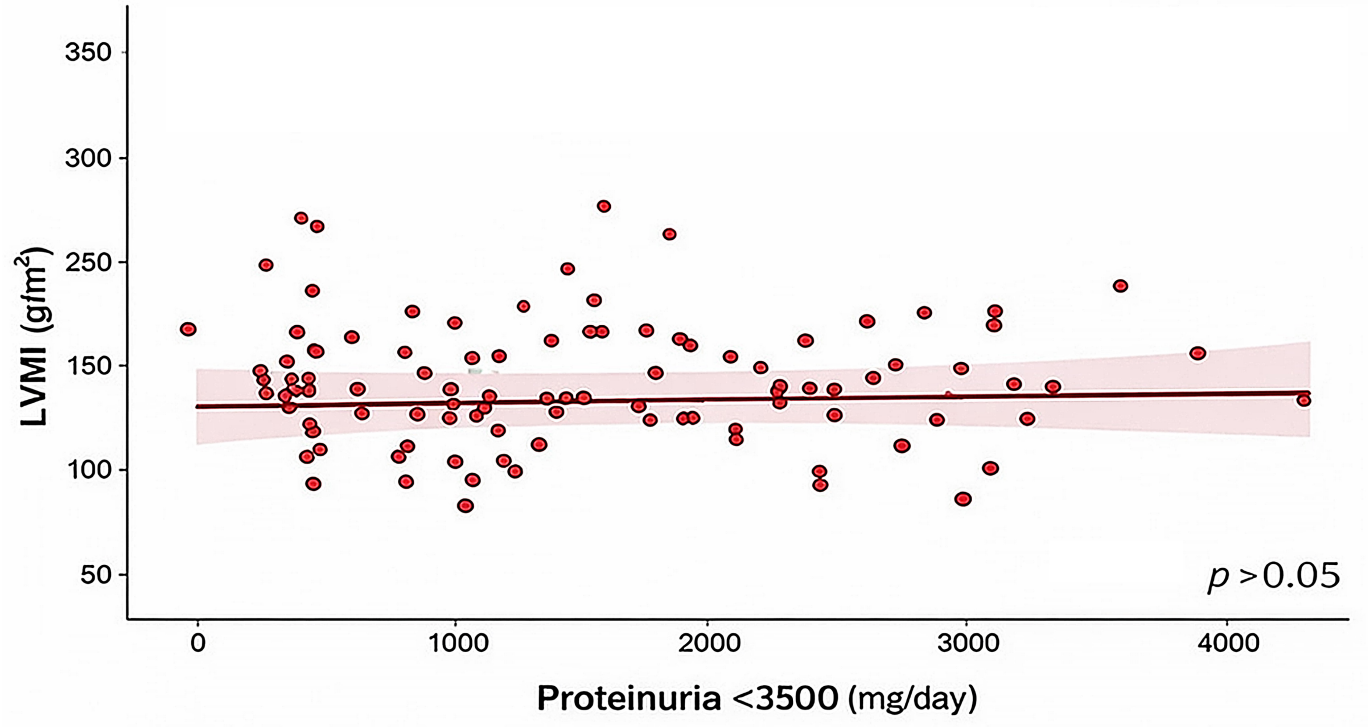

FIGURE 2. Relationship between non-nephrotic proteinuria and LVH. No relationship was found between non-nephrotic proteinuria and LVH ($p > 0.05$). LVMI: left ventricular mass index.

the association with LVH also increased. Wachtell *et al.* [26] reported that albuminuria in hypertensive individuals was associated with the development of LVH independent of blood pressure or the presence of diabetes. Nabbaale *et al.* [27] reported an association between microalbuminuria and LVH in 256 hypertensive patients. LVH was detected in 14.1% of the participants. They reported that microalbuminuria was associated with male gender, increased waist circumference, elevated blood pressure levels (systolic blood pressure ≥120 mmHg, diastolic blood pressure ≥100 mmHg), eGFR ≥50 mL/min/1.73 m^2, diabetes, congestive heart failure, cerebral pathology, and history of peripheral artery disease [27]. The authors reported that microalbuminuria in patients with GN is a risk factor for increased CV mortality and morbidity [1]. Murkamilov *et al.* [28], in a study conducted with 170 patients with chronic GN in the pre-dialysis CKD, reported a relationship between proteinuria and LVMI in males, whereas this relationship was not observed in females. In our study, increased LVH development was observed in patients with nephrotic proteinuria compared to those with non-nephrotic proteinuria among non-diabetic chronic GN patients. When examined according to the etiologies of GN, no difference was observed in the relationship between proteinuria and LVH across the different etiologies. An association between nephrotic proteinuria and LVH was found across all types of GN. This situation could have various underlying causes. The components of damage of glomeruli include the basement membrane, endothelial cells, and epithelial cells (podocytes), leading to protein loss in the urine. Due to the nephrotic-level protein loss in the urine, oncotic pressure decreases, leading to a reduction in circulating volume. This volume change stimulates the renin-angiotensin system. As a result, sodium and water retention occur, leading to hypertension and the development of LVH [29]. Another potential contributor could be the activation of the local renin-angiotensin aldosterone system, possibly mediated by fibroblast growth factor-23, which may contribute to the development of LVH by promoting myocardial hypertrophy and fibrosis [30]. Additionally, in chronic GN with nephrotic-level proteinuria, activation of autoimmune pathways leading to endothelial damage and low-grade inflammation may also contribute to the development of LVH and increased LVMI.

The results of this study show a significant association between physical activity level and the reduction in nephrotic proteinuria, left ventricular mass index (LVMI), and left ventricular hypertrophy (LVH) in patients with chronic kidney disease (CKD). These findings are consistent with previous research highlighting the positive effects of physical activity on kidney and cardiovascular health. It is well known that regular physical activity improves cardiovascular outcomes and reduces risk factors associated with chronic disease. In the current study, higher levels of physical activity were associated with significantly lower rates of proteinuria, which is consistent with the findings of Yang *et al.* [31], who reported that exercise interventions can improve proteinuria and cardiovascular outcomes in CKD patients. He *et al.* [32] indicated that regular physical activity is associated with improved cardiovascular health, but found limited direct effects on proteinuria in specific cohorts. In this study, both cardiovascular and renal benefits of increased physical activity were observed, as reflected by reduced LVMI and LVH rates. The observed decrease in LVMI and LVH with increased physical activity supports previous findings linking physical activity to improved cardiac remodeling and function. A possible explanation for the effects of physical exercise is

the modulation of myokines, which also have cardiovascular effects [33]. Studies such as that by Currie and Delles have shown that physical activity positively influences cardiovascular outcomes by reducing the burden of LVH, a key predictor of cardiovascular mortality in CKD patients [34]. However, several limitations of this study should be acknowledged. Self-assessment of physical activity may lead to bias, as patients may overestimate or underestimate their actual activity level. Furthermore, although the study design was robust, longitudinal data would provide stronger causal evidence for the observed associations.

5. Conclusions

An increased prevalence of LVH was observed in patients with chronic GN compared to healthy individuals. Similarly, a higher prevalence of LVH was observed in individuals with nephrotic proteinuria compared to individuals with non-nephrotic proteinuria. A significant association was found between proteinuria at the nephrotic level and LVH. However, due to the insufficient studies on this topic, there is a need for advanced multicenter studies with a larger number of patients. This study highlights the multiple potential benefits of physical activity in patients with CKD. It shows that it contributes to the reduction of nephrotic proteinuria, improves cardiac health, and leads patients to more favorable renal and cardiovascular outcomes. These findings provide strong support for incorporating tailored exercise programs into the clinical management of CKD patients, which is increasingly supported by a growing body of evidence. Future research should focus on prospective longitudinal studies to validate these findings and explore the mechanisms linking physical activity, kidney health, and cardiovascular outcomes.

Some limitations in our study may have influenced the results. First, because the study had a cross-sectional design, changes over time in the relationship between proteinuria and LVH in GN patients could not be evaluated. Second, proteinuria was measured using 24-hour urine collection, and the ratio of protein to creatinine in spot urine was not examined. Eknoyan *et al*. [35] reported a good correlation between the two methods in their study. Third, despite the known association of chronic GN with widespread vascular damage (microangiopathy), a pro-inflammatory state (elevated levels of interleukin-6, tumor necrosis factor-α, C-reactive protein, and fibrinogen), and subsequent endothelial dysfunction affecting the coronary arteries, inflammatory parameters were not evaluated in this study.

AVAILABILITY OF DATA AND MATERIALS

The data are available for research purposes upon reasonable request from corresponding author.

AUTHOR CONTRIBUTIONS

MİD and MÇ—designed the research study. BAD—performed the research. MŞK—provided help and advice on data interpretation. MİD—analyzed the data. MİD, MÇ, and BAD—wrote the manuscript. BB—supervised the study and provided critical revisions. All authors contributed to editorial changes in the manuscript. All authors read and approved the final version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Written informed consent was obtained from all participants after explaining the study to them. Approval number E2-22-1196 was received for the study from the Ethics Committee of Ankara Bilkent City Hospital.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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