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# ANALYSIS OF THE PREVALENCE AND ROLE OF MALADAPTIVE LEFT VENTRICULAR REMODELING IN THE RISK OF EARLY RENAL GRAFT DYSFUNCTION

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**Objective:** to study the prevalence of maladaptive left ventricular remodeling (MLVR) among kidney transplant (KT) candidates and the role of MLVR in the development of early graft dysfunction (EGD). **Materials and methods.** The study is based on a retrospective analysis of treatment outcomes in 650 patients who underwent a living related KT. Transthoracic echocardiogram revealed different types of left ventricular (LV) remodeling, whose prevalence was studied in the context of influence on the general population and specific "renal" risk factors. Two patient groups were also identified: Group I had EGD (n = 82) and Group II had primary graft function (PGF) (n = 79). These groups were comparable in terms of demographics, clinical data, and laboratory results ($p > 0.1$). The relative risk of developing EGD was calculated depending on whether maladaptive remodeling was present. **Results.** Concentric LV hypertrophy (cLVH) was detected in 341 (52.46%), eccentric (eLVH) in 174 (26.77%) patients. Concentric remodeling (CR) and normal LV geometry were detected in 86 (13.23%) and 49 (7.54%) patients, respectively. MLVR (cLVH + eLVH) was more common in men ($p = 0.003$). Compared to patients in the pre-dialysis stage, the risk of developing MLVR was 5.6 times higher for dialysis therapy durations up to 1 year, 8 times higher for durations 1 to 2 years, and 4.5 times higher for durations greater than 2 years ($p < 0.05$). The likelihood of developing MLVR was 8-fold higher in those with a functioning arteriovenous fistula ($p < 0.001$). As diuresis decreased, the odds of developing MLVR increased 4 to 15.8 times ($p < 0.001$). Depending on the severity of their anemia, patients with anemia had 2.7–13.8 times the chances of developing MLVR compared to those without anemia ($p < 0.05$). According to comparative analysis, the EGD group had a high prevalence of MLVR ($p = 0.01$). MLVR raised the risk of developing EGD in the post-transplant period by 8.5 times for cLVH ($p = 0.049$) and 14.5 times for eLVH ($p = 0.011$). **Conclusion.** The presence of MLVR in a KT candidate indicates the severity of cardiovascular disease brought on by progression of chronic kidney disease, and can also be regarded as one of the risk factors for EGD.

*Keywords: kidney transplantation, left ventricular hypertrophy, remodeling, early graft dysfunction.*

## INTRODUCTION

Today, kidney transplantation (KT) is widely considered the preferred method of replacement therapy for end-stage renal disease" (ESRD). It provides significantly better quality of life for patients and is considered more cost-effective compared to dialysis as a treatment for ESRD.

Cardiovascular health is a major determinant of life expectancy both in patients with chronic kidney disease (CKD) and in renal transplant recipients [1–3]. The diversity and close connection of pathological changes in kidney and cardiovascular damage led to the formation of the concept of cardiorenal syndrome (CRS), the definition and classification of which were first proposed by Ronco et al. in 2010 [4]. CRS is defined as a complex of pathological interdependent conditions involving the heart and kidneys, developing due to acute or chronic dysfunction in one of the organs, which leads to subsequent dysfunction in the other organ. In this article, we focus on CRS type 4, a condition where CKD leads to significant cardiovascular dysfunction.

In ESRD patients, the high risk of adverse cardiovascular events – including decompensated heart failure, arrhythmias, and acute coronary syndrome – is largely attributed to structural remodeling of the left ventricular (LV) myocardium. This remodeling results from increased LV mass, myocardial fibrosis, and geometric disturbances. Left ventricular hypertrophy (LVH) initially serves as a compensatory adaptation in early-stage CKD. However, as CKD progresses, LVH transforms into a pathological, maladaptive remodeling process [5].

In a clinical context, the progression from normal LV geometry to concentric remodeling (CR), then to concentric LV hypertrophy (cLVH) and eccentric LV hypertrophy (eLVH), represents distinct stages of maladaptive cardiac remodeling in CKD [6]. The high prevalence of

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cLVH in early CKD is primarily due to pressure overload, while the transition to eLVH at dialysis initiation is driven by volume overload and anemia [7]. In addition to hemodynamic causes, specific (renal) risk factors for LVH are common in CKD patients, with anemia having the greatest impact. Renal anemia in CKD primarily results from a deficiency of erythropoietin due to kidney damage. However, several additional factors worsen anemia in CKD, including iron deficiency and chronic inflammation.

LVH prevalence increases progressively from 16–31% in stage 1–3 CKD patients, reaching nearly 90% in dialysis-dependent patients. Meanwhile, CR is considered an adaptive form of LV remodeling, whereas cLVH and eLVH are classified as maladaptive left ventricular remodeling (MLVR) due to their strong association with adverse cardiovascular events (ACEs) [3, 5–9]. In a study by de Roij van Zuijdewijn CL et al. (2015), the risk of sudden death among patients with eLVH was 5.2 times higher than in the group of patients with cLVH and 10.2 times higher than in patients with normal LV geometry [10].

MLVR before KT is a strong predictor of ACEs and all-cause mortality in both the perioperative and long-term postoperative periods [11, 12].

KT can significantly improve cardiac function in most patients, but it takes time to trigger reverse remodeling processes [13, 14]. An experimental study by Hagmayer et al. (2023) demonstrated that KT in CKD rats led to improved cardiac function, but LVH persisted in most animals at 16 weeks post-transplant [15].

At the same time, decreased LV stroke volume, circulatory minute volume, cardiac index, increased total peripheral vascular resistance (PVR), arrhythmias, diastolic and systolic dysfunction caused by MLVR may have an adverse effect on kidney graft function in the early post-transplant period.

Thus, LVH prevalence among KT candidates is a crucial area of investigation due to its direct impact on transplant outcomes, particularly the risk of early graft dysfunction. We found no studies addressing this issue in available scientific databases.

**Objective:** to investigate MLVR prevalence among KT candidates, to determine its significance in the development of early graft dysfunction (EGD).

## MATERIALS AND METHODS

The study is based on a retrospective analysis of the treatment outcomes of 650 patients with ESRD who underwent a living-related kidney transplantation (LRKT) at Vakhidov Republican Specialized Research and Practical Medical Center of Surgery from January 2018 to August 2022.

Patients were included in the study using a continuous method. Inclusion criteria for the study were stage 5 CKD and LRKT. The sample did not include patients who did not have a histocompatible donor or were not allowed for transplantation due to chronic underlying diseases in the decompensation stage.

The patients were examined at the outpatient and hospital stage of preparation for KT surgery according to the approved national protocol for examining KT candidates (Order of the Ministry of Health of the Republic of Uzbekistan, No. 179 dated June 27, 2022, Appendix No. 2 “List of tests for medical examination of a living donor and recipient”).

A donor-recipient pair was selected taking into account histocompatibility on the basis of HLA I–II analysis; lymphocytotoxic test was also performed. Determination of markers of hepatitis B, C, HIV, TORCH complex, biochemical and hematological studies were performed at the laboratory of Vakhidov Republican Specialized Research and Practical Medical Center of Surgery in Tashkent, Uzbekistan, on automatic analyzers BC-5300 (Mindray, China), Vitros-350 (G&G, USA), and Maglumi-800 (China).

An echocardiogram was performed on ultrasound scanners GE LOGIQ P6 (General Electric Health Care, USA), Philips HD11 XE (Philips Healthcare, USA), and Toshiba Xario 200 (Toshiba Medical Systems Corp., Japan) using 3–5 MHz phased array probes. A standard transthoracic echocardiogram (TTE) was performed according to the guidelines of the American Society of Echocardiography [16]. In patients receiving hemodialysis sessions, the study was performed mainly on the day after hemodialysis procedure, thus leveling the factor of volume overload associated with interdialysis increase in the volume of extracellular fluid.

As a result of measurements from parasternal, apical, subcostal accesses, in M-, B-modes, structural-geometric parameters, systolic heart function parameters, assessment of the valve apparatus state (using pulsed-wave, constant-wave Doppler mode) were determined.

Linear parameters were studied in M-mode according to the standard Penn convention method, and the following were determined: left ventricular end-diastolic diameter (LVEDD, cm); left ventricular end-systolic diameter (LVESD, cm); interventricular septum thickness at systole and diastole (IVSs, IVSd, cm); left ventricular posterior wall thickness at systole and diastole (LVPWs, LVPWd, cm). The LV relative wall thickness (RWT) was calculated using the formula:

$$RWT = 2 \times LVPWd / LVEDD.$$

Based on the obtained data, left ventricular myocardial mass (LVMM) was calculated using the formula developed by Devereux et al. (1986), recommended by the American Society of Echocardiography (ASE), based on linear measurements and the LV model as an elongated ellipsoid of revolution:

$$LVMM = 0.8 \times (1.04 \times [(EDD + LVPWd + IVSd)^3 - (EDD)^3]) + 0.6 \text{ gr.}$$

The result was weighed against body surface area (BSA) in $m^2$ to obtain the LVMM. BSA was calculated using the Gehan & George method in accordance with European Renal Best Practice Guideline on kidney donor and recipient evaluation and perioperative care [17]:

$$BSA\ (m^2) = 0.0235 \times \text{growth (cm)}^{0.42246} \times \times \text{weight (kg)}^{0.51456}.$$

An LVMM of more than 115 g/$m^2$ in men and 95 g/$m^2$ in women was considered as LVH criterion according to ASE and European Association of Echocardiography (EAE) guidelines. Types of LV hypertrophy and remodeling were determined depending on LVMM and RWT according to the classification proposed by Antonello Ganau (1992):

- normal geometry (normal LVMM; RWT ≤0.42)
- concentric remodeling (normal LVMM; RWT >0.42)
- concentric hypertrophy (increased LVMM; RWT >0.42)
- eccentric hypertrophy (increased LVMM and RWT ≤0.42).

Primary graft function (PGF) was observed in 539 (82.92%) patients. Delayed graft function (the need for dialysis within the first 7 days after KT) was observed in 48 (7.38%) patients. Delayed graft function was determined by a 2-fold increase in plasma creatinine within the first 5 days after operation, and was observed in 34 (5.23%) patients. Acute renal graft rejection was observed in 9 (1.38%) cases, and infectious and surgical complications leading to graft dysfunction or loss were observed in 12 (1.85%) cases.

Patients with delayed graft function were grouped into study group I (n = 82) with early graft dysfunction (EGD). For comparison, group II (n = 79), comparable in terms of clinical and demographic parameters, was selected among patients with PGF (n = 539).

Statistical processing was carried out using parametric and nonparametric analysis methods. Accumulation, adjustment, systematization of initial information and visualization of the obtained results were done in Microsoft Office Excel 2016 spreadsheets. Statistical analysis was performed using the IBM SPSS Statistics v.26 program developed by IBM Corporation, USA.

Conformity of quantitative indicators to normal distribution was measured using the Kolmogorov–Smirnov test. In the case of describing the indicators having normal distribution, the obtained data were presented in the form of arithmetic mean (M) and standard deviation (SD); for indicators that did not follow a normal distribution, data were presented as median (Me) with lower and upper quartiles (Q1; Q3). Nominal data were described with absolute values and percentages.

When comparing mean values in normally distributed sets of quantitative data, the Student t-test was calculated; in cases of no signs of normal distribution, we used Mann–Whitney U test.

Nominal data were compared using Pearson's chi-squared test; where the expected phenomenon was less than 10, we calculated the chi-squared test with a Yates correction to reduce the probability of type 1 error.

As a quantitative measure of effect when comparing relative measures, we used the odds ratio (OR), defined as the ratio of the probability of an event occurring in the exposed group to the probability of the event occurring in the control group. In order to project the obtained OR values to the general population, we calculated the boundaries of the 95% confidence interval (95% CI). Statistical significance of differences was recognized at a significance level of $p < 0.05$.

## RESULTS

The median age of the study cohort was 33 (27–39) years, with a predominance of young people (18–44 years), n = 543 (83.54%). In terms of gender composition, males predominated – 476 (73.23%) versus 174 (26.7%) females. Body mass index (BMI) was calculated using the formula: body weight (kg) / height ($m^2$). Median BMI was 22.7 kg/$m^2$ (20.2; 25.3). Renal replacement therapy by long-term hemodialysis was received by 565 (86.92%) patients. Median duration of CKD was 24 (12; 50) months and median length of dialysis was 9 (5; 16) months. Vascular access was via arteriovenous fistula (AVF) (n = 507; 89.73%) or central venous catheter (CVC) (n = 58; 10.27%). In 85 (13.08%) cases, KT was performed in the pre-dialysis stage of the disease.

The structure of clinical entities of renal diseases that were the cause of ESRD in our study is presented in Fig. 1. In most cases, the cause of ESRD in the studied patient cohort was chronic glomerulonephritis (n = 554; 85.23%). Among other pathology: 3 (0.46%) cases each of diabetic and gouty nephropathy; 2 (0.31%) cases each of neurogenic bladder, interstitial nephritis in pregnancy, lupus nephritis; one patient was diagnosed with Alport syndrome, a rare hereditary pathology of basal membranes, which is manifested by hematuria and progressive decline in renal function.

When studying the prevalence of various forms of LV remodeling in the studied patient cohort, it was revealed that the most common form was cLVH, detected in 341 (52.46%) patients. It is also worth noting the relatively high prevalence of the most unfavorable form of remodeling – eLVH (n = 174; 26.77%). Normal LV geometry was detected only in 49 (7.54%) patients (Fig. 2).

Table 1 presents the results of analysis of the influence of traditional (gender, age, BMI) and renal (length of dialysis therapy, type of vascular access, residual diuresis, renal anemia) risk factors on the prevalence of maladaptive remodeling (cLVH + eLVH).

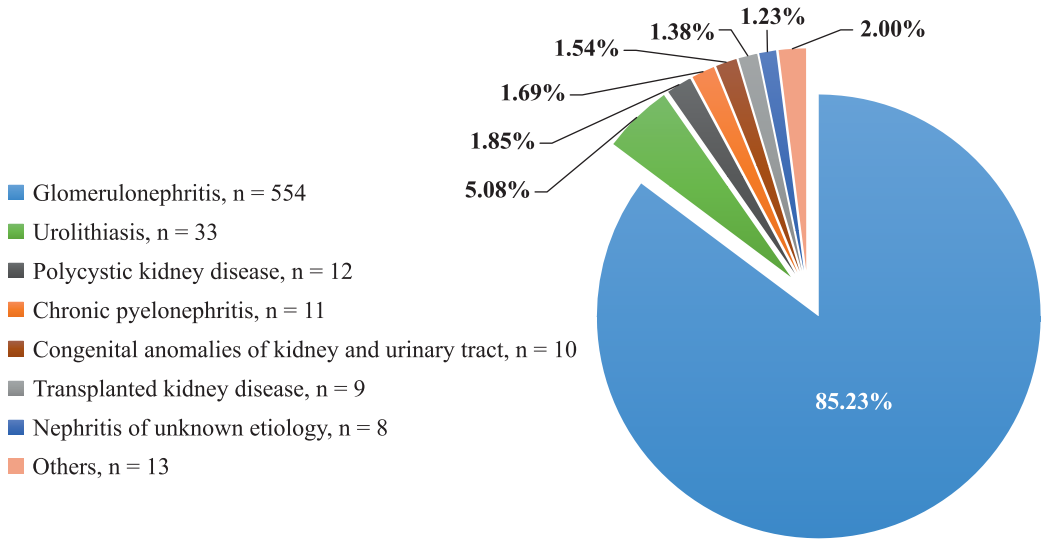


Fig. 1. Classification of kidney diseases

Table 1

**Factor analysis of the prevalence of maladaptive left ventricular remodeling among kidney transplant candidates**

| | Patients with maladaptive remodeling (cLVH + eLVH) | | | | | | Normal geometry | |
|---|---|---|---|---|---|---|---|---|
| | n | % | $\chi^2$ | p | OR | 95% CI | n | % |
| Entire cohort (n = 650) | 515 | 79.2 | | | | | 49 | 7.5 |
| **Sex** | | | | | | | | |
| Male (n = 476) | 395 | 83 | 9.13 | 0.003 | 2.469 | 1.353–4.505 | 28 | 5.9 |
| Female (n = 174) | 120 | 69 | | | | | 21 | 12.1 |
| **Age** | | | | | | | | |
| ≤20 years (n = 37) | 20 | 54.1 | | | | | 7 | 18.9 |
| 21–44 years (n = 524) | 431 | 82.3 | 9.686 | 0.002 | 4.571 | 1.802–11.594 | 33 | 6.3 |
| 45–59 years (n = 70) | 50 | 71.4 | 2.143 | 0.144 | 2.917 | 0.872–9.756 | 6 | 8.6 |
| 60–74 years (n = 19) | 14 | 73.7 | 0.072 | 0.789 | 1.633 | 0.359–7.432 | 3 | 15.8 |
| **BMI** | | | | | | | | |
| BMI < 18.4 (n = 86) | 62 | 72.1 | 0.237 | 0.627 | 0.722 | 0.300–1.737 | 7 | 8.1 |
| BMI 18.5–24.9 (n = 385) | 319 | 82.9 | | | | | 26 | 6.8 |
| BMI 25–29.9 (n = 139) | 108 | 77.7 | 0.157 | 0.692 | 0.8 | 0.383–1.674 | 11 | 7.9 |
| BMI >30 (n = 40) | 26 | 65 | 1.757 | 0.186 | 0.424 | 0.150–1.196 | 5 | 12.5 |
| **Duration of dialysis** | | | | | | | | |
| ≤12 months (n = 364) | 308 | 84.6 | 23.473 | 0.001 | 5.600 | 2.717–11.543 | 22 | 6.0 |
| 1–2 years (n = 122) | 100 | 82 | 16.215 | 0.001 | 8.000 | 2.747–23.301 | 5 | 4.1 |
| >2 years (n = 79) | 67 | 84.8 | 7.896 | 0.005 | 4.467 | 1.616–12.347 | 6 | 7.6 |
| Pre-dialysis (n = 85) | 40 | 47.1 | | | | | 16 | 18.8 |
| **Vascular access** | | | | | | | | |
| AVF (n = 507) | 440 | 86.8 | 38.223 | 0.001 | 8.000 | 3.891–16.448 | 22 | 4.3 |
| CVC (n = 58) | 35 | 60.3 | 0.282 | 0.596 | 1.273 | 0.522–3.105 | 11 | 19.0 |
| **Residual urine output** | | | | | | | | |
| Anuria (n = 93) | 86 | 92.5 | 21.575 | 0.001 | 15.809 | 3.617–69.101 | 2 | 2.2 |
| ≤500 mL/day (n = 304) | 264 | 86.8 | 34.828 | 0.001 | 7.466 | 3.630–15.358 | 13 | 4.3 |
| 500–1500 mL/day (n = 129) | 97 | 75.2 | 11.828 | 0.001 | 3.962 | 1.741–9.020 | 9 | 7.0 |
| >1500 mL/day (n = 124) | 68 | 54.8 | | | | | 25 | 20.2 |
| **Renal anemia** | | | | | | | | |
| Mild (n = 193) | 138 | 71.5 | 7.276 | 0.007 | 2.684 | 1.288–5.595 | 19 | 9.8 |
| Moderate (n = 255) | 219 | 85.9 | 27.483 | 0.001 | 8.093 | 3.482–18.810 | 10 | 3.9 |
| Severe (n = 121) | 112 | 92.6 | 21.866 | 0.001 | 13.797 | 3.857–49.350 | 3 | 2.5 |
| No anemia (n = 81) | 46 | 56.8 | | | | | 17 | 21.0 |

*Note:* cLVH, concentric left ventricular hypertrophy; eLVH, eccentric left ventricular hypertrophy; BMI, body mass index; AVF, arteriovenous fistula; CVC, central venous catheter.

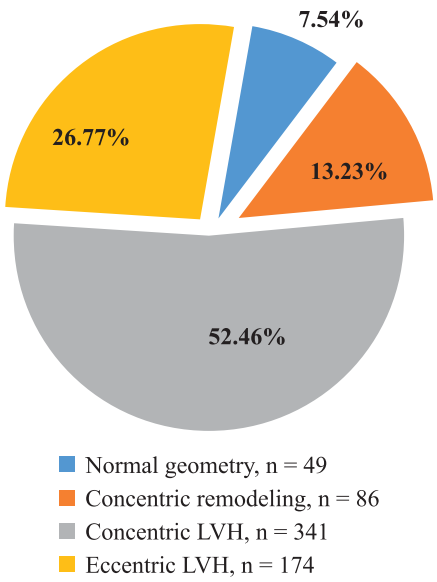


Fig. 2. Prevalence of left ventricular remodeling types

As shown in the presented table, male gender was associated with a high chance of developing cLVH and eLVH (OR 2.469; CI 95% 1.353–4.505; p = 0.003). Patient age and weight had no significant effect. Renal risk factors had a significant impact on the prevalence of maladaptive remodeling. For example, the odds of developing cLVH and eLVH were 5.6 times (p < 0.001) higher for dialysis lasting for less than 1 year, 8 times (p < 0.001) for 1 to 2 years, and 4.5 times (p = 0.005) for more than 2 years compared to pre-dialysis patients. The presence of a functioning AVF was associated with an 8-fold increase in the odds of developing cLVH and eLVH (p < 0.001) compared to pre-dialysis patients, where the influence of shunt blood flow was absent. We also found that progressive decrease in diuresis significantly increased the odds of maladaptive remodeling by 4 to 16 times (p < 0.001) compared to patients with preserved diuresis (>1500 mL/day). We consider renal anemia as a specific renal risk factor, but it is worth noting that anemia is also a risk factor for LVH in the general population [8]. The chances of developing MLVR in persons with anemia, depending on its degree, are 2.7 to 13.8 times (p < 0.001) higher than in the anemia-free group.

For the next stage of the study, we selected a group of PGF patients comparable in terms of the main clinical and demographic parameters in comparison with the EGD patient group; the obtained data are presented in Table 2.

The absence of statistically significant differences in the presented parameters allowed us to level their potential influence on the prevalence of various forms of remodeling and graft function between the study groups. This condition was necessary to study the possible influence of MLVR on the risk of early renal graft dysfunction.

A comparative analysis of transthoracic echocardiogram results between the study groups is presented in Table 3.

The presented results suggest that patients who developed EGD exhibited distinct cardiac abnormalities on preoperative echocardiography (EchoCG), including LV dilatation, reduced myocardial contractility, severe

Table 2

**Comparative analysis by primary clinical and demographic indicators**

| | Group I (n = 82) (EGD) | | Group II (n = 79) (PGF) | | p |
|---|---|---|---|---|---|
| Age (Me; Q1; Q3) | 32.5 | 27; 38 | 33 | 26; 37.5 | 0.907 |
| Male (n/%) | 66 | 80.49% | 63 | 79.75% | 0.908 |
| Female (n/%) | 16 | 19.51% | 16 | 20.25% | |
| BMI (M ± SD) | 22.99 | 3.41 | 22.51 | 3.41 | 0.384 |
| **Duration of dialysis** | | | | | |
| Pre-dialysis (n/%) | 6 | 7.32% | 7 | 8.86% | |
| ≤12 months (n/%) | 51 | 62.20% | 41 | 51.90% | 0.741 |
| 12–24 months (n/%) | 17 | 20.73% | 15 | 18.99% | 0.925 |
| >24 months (n/%) | 8 | 9.76% | 16 | 20.25% | 0.445 |
| **Residual urine output** | | | | | |
| Anuria (n, %) | 18 | 21.95% | 17 | 21.52% | 0.964 |
| ≤500 mL/day (n, %) | 41 | 50.00% | 34 | 43.04% | 0.904 |
| 500–1500 mL/day (n, %) | 15 | 18.29% | 22 | 27.85% | 0.455 |
| >1500 mL/day (n, %) | 8 | 9.76% | 6 | 7.59% | |
| **Renal anemia** | | | | | |
| No anemia (n, %) | 8 | 9.76% | 10 | 12.66% | |
| Mild (n, %) | 22 | 26.83% | 19 | 24.05% | 0.713 |
| Moderate (n, %) | 26 | 31.71% | 28 | 35.44% | 0.786 |
| Severe (n, %) | 26 | 31.71% | 22 | 27.85% | 0.670 |

*Note:* EGD, early graft dysfunction; PGF, primary graft function; BMI, body mass index.

left LVH, increased prevalence of mitral and tricuspid regurgitation, and severe pulmonary hypertension.

The comparative prevalence of maladaptive remodeling in the study groups is presented in Fig. 3. Differences between groups evaluated using Pearson's chi-squared test were statistically significant ($\chi^2 = 11.497$; $p = 0.01$), which allowed us to conclude that cLVH and eLVH are highly prevalent in the group of patients with EGD.

Quantitative analysis of the effect of MLVR on EGD demonstrated that the presence of cLVH increased the odds of EGD 8.5-fold (95% CI: 1.027–71.134; p = 0.049), while eLVH increased the odds 14.5-fold (95% CI: 1.661–126.57; p = 0.011). In contrast, CR did not have a statistically significant effect on EGD development (Table 4).

## DISCUSSION

Our study revealed that MLVR was highly prevalent (79.23%) among the study population, with cLVH being the most common subtype (52.46%). These findings align with previously reported data, indicating that LVH is present in 48–84% of patients with CKD at the

Table 3

**Comparative analysis of transthoracic echocardiogram results in patients depending on kidney graft function**

| | Group I (n = 82) (EGD) | | Group II (n = 79) (PGF) | | p |
|---|---|---|---|---|---|
| EDV, mL (M ± SD) | 170.74 | 41.97 | 154.51 | 44.76 | 0.016 |
| ECV, mL (Me; Q1; Q3) | 79 | 59; 99 | 64 | 46.5; 89 | 0.002 |
| SV, mL (M ± SD) | 87.77 | 23.42 | 84.70 | 21.82 | 0.362 |
| EF % (M ± SD) | 51.35 | 10.45 | 56.05 | 8.08 | 0.002 |
| IVSd, cm (Me; Q1; Q3) | 1.5 | 1.3; 1.8 | 1.4 | 1.2; 1.6 | 0.060 |
| LVPWd, cm (Me; Q1; Q3) | 1.5 | 1.3; 1.8 | 1.4 | 1.2; 1.65 | 0.058 |
| RWT (Me; Q1; Q3) | 0.52 | 0.41; 0.6 | 0.52 | 0.42; 0.62 | 0.450 |
| LVMM (Me; Q1; Q3) | 245.13 | 184.79; 325.33 | 197.28 | 150.49; 265.95 | 0.001 |
| EDD (M ± SD) | 5.92 | 0.87 | 5.39 | 0.96 | <0.001 |
| ESD (Me; Q1; Q3) | 4 | 3.5; 4.5 | 3.7 | 3.3; 4.2 | 0.009 |
| MR II, III (n, %) | 47 | 57.32% | 28 | 35.44% | 0.006 |
| TR II, III (n, %) | 41 | 50.00% | 15 | 18.99% | <0.001 |
| AR II (n, %) | 3 | 3.66% | 1 | 1.27% | 0.640 |
| mPAP, mmHg (M ± SD) | 48.26 | 18.63 | 31.92 | 16.11 | <0.001 |

*Note:* EDV, end-diastolic volume; ECV, end-systolic volume; SV, stroke volume; EF, ejection fraction; IVSd, interventricular septum thickness at diastole; LVPWd, left ventricular posterior wall thickness at diastole; RWT, relative wall thickness; LVMM, left ventricular myocardial mass; EDD, end-diastolic diameter; ESD, end-systolic diameter; MR, mitral regurgitation; TR, tricuspid regurgitation; AR, aortic regurgitation; mPAP, mean pulmonary artery pressure.

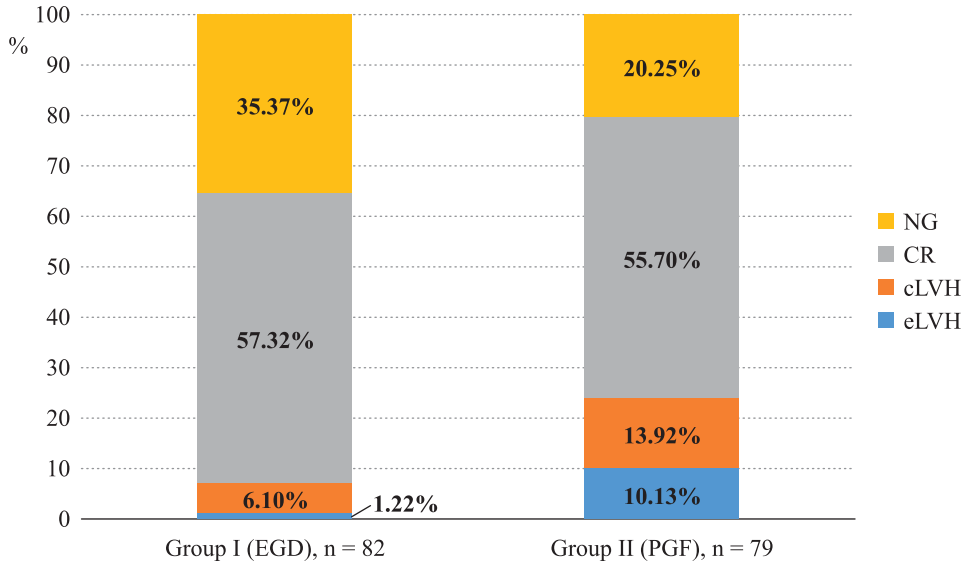


Fig. 3. Prevalence of left ventricular remodeling types in the study groups. NG, normal geometry; CR, concentric remodeling; cLVH, concentric LV hypertrophy; eLVH, eccentric LV hypertrophy; EGD, early graft dysfunction; PGF, primary graft function

Table 4

**Risks of developing early allograft dysfunction depending on types of left ventricular remodeling**

| | Group I (n = 82) (EGD) | Group II (n = 79) (PGF) | $\chi^2$ | p | OR | 95% CI |
|---|---|---|---|---|---|---|
| Normal LV geometry | 1 (1.22%) | 8 (10.13%) | | | | |
| Concentric remodeling | 5 (6.1%) | 11 (13.92%) | 1.281 | 0.52 | 3.64 | 0.353–37.458 |
| Concentric LV remodeling | 47 (57.32%) | 44 (55.7%) | 3.89 | 0.049 | 8.55 | 1.027–71.134 |
| Eccentric LV remodeling | 29 (35.37%) | 16 (20.25%) | 6.615 | 0.011 | 14.50 | 1.661–126.570 |

*Note:* EGD, early graft dysfunction; PGF, primary graft function; OR, odds ratio; LV, left ventricular.

pre-dialysis stage and up to 90% of patients on long-term hemodialysis [7–9].

Among the identified risk factors, male gender ($p = 0.003$), longer duration of dialysis therapy ($p < 0.001$), presence of a functioning AVF ($p < 0.001$), decreased diuresis ($p < 0.001$), and renal anemia ($p < 0.001$) were significantly associated with a higher prevalence of MLVR. Information about the influence of the above factors on MLVR development according to reports is contradictory, except for renal anemia, whose role in the development of LVH has been confirmed by most studies [5–9, 18, 19]. In addition, renal anemia is a modifiable risk factor for MLVR, and its effective treatment may significantly promote LVH regression [20]. Evidence also suggests that in ESRD patients, anemia has a greater influence on myocardial remodeling than blood pressure [18].

We investigated the prevalence of various forms of LV remodeling among KT candidates, particularly in relation to their potential impact on transplant outcomes, including the risk of EGD. Our findings indicate that the likelihood of developing EGD is 8.5-fold higher in cLVH and 14.5-fold higher in eLVH compared to patients with normal LV geometry. These results suggest that patients with MLVR should be considered at increased risk for EGD.

KT eliminates the need for long-term dialysis and improves renal function, which contributes to regression of LVH and other structural cardiac changes. These improvements ultimately enhance overall survival and quality of life in transplant recipients. Despite advancements in modern transplantology, EGD remains a concern, occurring in approximately 15–30% of cadaveric transplants and 5–10% of living donor transplants. Various donor- and recipient-related risk factors for EGD are still under investigation. Similarly, EGD significantly impacts long-term graft survival in the postoperative period [21–23].

Based on both literature data and our own research findings, this relationship underscores the critical importance of eliminating modifiable risk factors for LVH, particularly symptomatic hypertension and renal anemia. Effective control of these factors can significantly reduce the risk of MLVR development and progression, ultimately leading to improved immediate post-transplant renal outcomes. In addition, evidence suggests that optimizing dialysis therapy – specifically, achieving the patient's dry weight and maintaining interdialytic weight gain below 5% of body weight – can contribute to LVH regression [19].

A promising area for further investigation is the role of the endogenous cardiotonic steroid marinobufagenin (MBG) in LVH development and progression in CKD patients, including those who have undergone KT. A study by Bolignano et al. demonstrated that MBG levels in KT recipients are lower than those observed in hemodialysis patients but remain elevated compared to healthy individuals. MBG levels were found to directly correlate with left ventricular myocardial mass and serve as a significant predictor of post-KT cardiovascular and renal complications. The authors proposed considering MBG as a unifying factor in pathological cardiac remodeling and kidney graft dysfunction. Its measurement holds significant prognostic value in enhancing cardiorenal risk assessment [24].

## CONCLUSION

The identification of MLVR in KT candidates is a crucial component of preoperative comorbidity assessment. Early detection can help reduce the risk of postoperative complications, including EGD. Further investigation into the impact of MLVR on immediate KT outcomes necessitates multicenter randomized clinical trials to provide robust evidence.

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