Risk factors for global longitudinal strain impairment in renal transplant recipients with preserved left ventricular ejection fraction

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Abstract

Background. Patients with chronic kidney disease, including those after kidney transplantation (KTx), have higher cardiovascular mortality. Global longitudinal strain (GLS) detects subtle changes in the left ventricle (LV) and is more sensitive predictor of cardiovascular mortality than the LV ejection fraction (LVEF). The aim of this study was to assess the prevalence of impaired GLS among kidney transplant recipients with preserved LVEF. We also aimed to identify possible clinical factors responsible for GLS impairment. Methods. A total of 79 patients following KTx with preserved LVEF and no history of cardiac disease were evaluated. We assessed echocardiogram parameters with the calculation of GLS, laboratory parameters, presence of diabetes, hypertension, duration of haemodialysis (HD) and time after KTx. An impaired GLS value was set on ≥-18%. The Classification and Regression Trees and stepwise logistic regression analysis were used to identify the factors related to impaired GLS. Results. Among 79 (42 females, mean age 60.3) kidney transplant recipients with preserved LVEF, 39% had impaired GLS. In stepwise logistic regression analysis, two variables related with GLS≥-18%: duration of HD before KTx longer than 27.5 months (OR 4.48, 95% CI 1.46-14.38, P=0.01), and eGFR greater than 60mL/min/1.73m2 (OR 0.155, 95% CI 0.024-0.967, P= 0.049). Conclusions. In our study group, a total of 39% of KTx patients with preserved LVEF had impaired GLS. The main risk factor of GLS impairment was the prolonged time of HD (>27.5 months) prior to KTx, whereas a good renal graft function (eGFR>60mL/min/1.73m2) only marginally proved to be a protective factor.

Background

Cardiovascular disease is the major threat for survival of patients with chronic kidney disease (CKD) including those after kidney transplantation (KTx). The risk of cardiovascular disease rises by 15% for every increasing stage of CKD [1]. The CKD from stage 3 to the end-stage renal disease (ESRD) is associated with volume and pressure overload as well as with renal specific risk factors for cardiomyopathy leading to left ventricular hypertrophy (LVH), reduced capillary density, and increased myocardial fibrosis.

During dialysis treatment abnormal myocardial remodelling and LVH progress due to additional
factors such as bone mineral disorders, inflammation, oxidative stress, arterio-venous fistula, and many others. The LVH is observed at about 75% of adult patients and 69% of paediatric patients after the initiation of dialysis, and among almost all patients after 5 years on dialysis therapy [2,3,4]. LVH and increased myocardial fibrosis are causes of coronary disease, heart failure, arrhythmia and sudden cardiac death.

Successful KTx improves long-term survival in comparison to dialysis patients. However, the risk of cardiovascular disease is still three- to five-fold higher in comparison to the age-matched general population [5].

KTx reduces some of the factors of cardiomyopathy, such as volume and pressure overload, and uremia; but immunosuppressive treatment based on calcineurin inhibitors and steroids contributes to hypertension and metabolic disorders. Moreover, most of the kidney transplant recipients have renal impairment and are in stage 3 CKD according to KDIGO (Kidney Disease: Improving Global Outcomes). CKD patients, including kidney transplant patients, with preserved left ventricle ejection fraction (LVEF), and with no symptoms of cardiovascular disease have subclinical left ventricle (LV) systolic dysfunction, which is not detected in the standard echocardiography. Detection of a subtle damage of LV systolic contractility is possible by speckle-tracking echocardiography using global longitudinal strain (GLS).

GLS is a percentage change in length of myocardial segment in systole and is presented as negative value. The prognostic value of impaired GLS of LV is well documented among patients with various cardiovascular diseases. In recent years some studies have confirmed impaired GLS as a predictor of all-cause and cardiovascular mortality among patients with CKD [6,7]. So far, there have been no studies on clinical factors affecting GLS impairment in kidney transplant recipients.

The aim of the study was to assess the prevalence of impaired GLS among kidney transplant recipients with preserved LVEF and to identify the clinical factors for reduced GLS in these patients.

Methods

**Patient population**

We performed a retrospective analysis of 79 adult patients after a successful KTx. All patients
underwent the echocardiography between 2015-2018. Our study included only stable patients with sinus rhythm and preserved LVEF, without moderate or severe valve pathology and coronary artery disease. Patients' demographic and clinical data were extracted from the patient's electronic medical records and then verified with paper charts. The collected variables relate to the date of the echocardiographic examination. Demographic and clinical data included: age, gender, body mass index (BMI), body surface area (BSA), presence of diabetes (DM), hypertension (HTN), duration of haemodialysis (HD) before and after KTx. Laboratory variables included both serum lipid concentration and assessment of renal graft function (e.g. serum creatinine, estimated glomerular filtration rate (eGFR), and uric acid).

**Echocardiography**

Transthoracic echocardiography examination was performed for each patient for clinical indications using a high-resolution ultrasound machine (*GE Vivid E9, Horten, Norway*) in accordance with the European Society for Echocardiography recommendations. All measurements and STE analysis was performed by an experienced echocardiographer (O.M). The linear measurements of the left ventricle, such as LV end-diastolic diameter (LVEDd), intraventricular end-diastolic diameter (IVDd), posterior end-diastolic wall thickness diameter (PWTd), and left atrium diameter (LAd) were obtained by using the M-mode ultrasound imaging. The LV mass (LVM) was estimated according to the equation below and then indexed to the BSA.

\[
LV \text{ Mass [g]} = 0.8 \left( 1.04 \left( \frac{[LVEDD + IVSd + PWd]^3 - LVEDD^3]}{LVEDD} \right) \right) + 0.6
\]

The upper limit of the LV mass index (LVMI) was set on 115g/m² for men and 95g/m² for women. Severe LVH was considered as LVMI≥149/m² and ≥122/m² for men and women, respectively. The relative wall thickness (RWT) was calculated for classified LV geometry, using the following formula:

\[
RWT = \frac{2PWTd}{LVEDd}
\]

The concentric LVH was diagnosed in the presence of the following factors: increased LVMI, normal LV size and RWT>0.42, whereas the eccentric LVH was diagnosed in the presence of an increased LVMI, increased LV size, and RWT≤0.42. The LVEF was measured using the modified Simson’s rule. The Doppler echocardiography was used to evaluate the diastolic function of
LV such as the early diastolic mitral inflow (E) and late diastolic mitral inflow (A) in order to derive E/A ratio and deceleration time. Doppler tissue imaging was performed in a four-chamber view to measure lateral and septal velocities of the mitral annulus, such as the peak early diastolic filling velocity (e) and the peak late diastolic filling velocity (a). Therefore, the ratio of E to average e (lateral+septal/2) was evaluated. GLS was measured using a two-dimensional STE and was then provided by the software as the average peak systolic longitudinal strain value of the three apical views and expressed as a negative value. The normal value of GLS was considered below -18%. [8]. Basically, the more negative the GLS value, the better cardiac function. Additionally, our study population was divided into two groups according to LV GLS < -18% (with normal GLS) and ≥-18% (with impaired GLS).

**Statistical analysis**

Continues variables were presented as mean and standard deviation and were compared with Student’s T-test or Mann Whitney U test as appropriate. The discrete variables were presented as noun and percentages and were compared with the Chi-square test with Yates correction when indicated. Classification and Regression Trees (CART) analysis were used to identify cut-off points for a duration time of HD and time which elapsed after KTx to predict GLS ≥-18%. The CART is a non-parametric statistical method. It is used to identify predictor variables using the method of binary partitioning. The statistical program uses each possible value of each independent variable to find cut off points. The CART generates a classification rule. This rule is presented as “a classification-tree”. This method shows the relationship between an assessed outcome (GLS≥-18%) and taken into account independent variables. The graph (classification trees) consists of rectangles. Every rectangle is numbered. The distinctive number is positioned in the left upper corner. In the right corner, there is a number referring to the outcome prevalence in the given subgroup. Finally, the legend is placed in the top left corner of the graph. When two lines are drawn from the rectangle’s bottom to connect to another two rectangles (child nodes). The child nodes differ in term of outcome prevalence. The text between the two lines describes the split. The numbers (N=) represent the number of cases directed into the child nodes. The accuracy of a global 10-fold cost validation of the
The multivariate stepwise logistic regression analysis was performed to find independent variables related to GLS ≥ -18%. The factors which in univariate analysis differ between groups with \( P \) less than 0.15, and variables of clinical significance were used as independent variables.

A \( P \)-value of less than 0.05 was regarded as statistically significant.

Results

A total of 79 (53% females) kidney transplant recipients at mean (SD) age 60.3 (11.5) were included in the study. Among the reasons of KTx were: glomerulonephritis in 27 (34.6%) patients, polycystic kidney disease in 16 (20.5%), HTN in 12 (15.4%), nephrolithiasis in 2 (2.6%), carcinoma in 1 (1.3%), kidney defects in 6 (7.7%), DM in 3 (3.9%), acute renal insufficiency in 1 (1.35%), interstitial nephritis in 1(1.3%), and the cause was unknown in 9 (11.5%) patients.

The average (SD) duration of renal replacement therapy was 144 (80.6) months and included the time of HD before KTx, and time from KTx up to the day the echocardiography examination was performed. The mean (SD) time after KTx was 120.2 (71.5) months, while the mean (SD) time of HD before KTx was 23.4 (23.4) months. Ten patients did not undergo HD before KTx.

Patients were divided into two groups depending on the GLS value. Group 1 consisted of 31 patients with GLS ≥ -18%, whereas Group 2 consisted of 48 patients with GLS < -18%. The demographic and clinical characteristic of the two groups is shown in Table 1. Both groups were similar in terms of age, women's presence and BMI, whereas BSA was significantly higher in Group 1 (\( P = 0.027 \)).

There were no significant differences between groups in the incidence of DM, HTN. The laboratory results were similar in both groups and are presented in Table 2. The percentage of patients with eGFR above 60mL/min/1.73m\(^2\) was similar in both groups. All patients had preserved LVEF.

Echocardiographic parameters in study groups are shown in Table 3

The LAd measured in from the parasternal long axis view was significantly higher in Group 1 (\( P = 0.006 \)) but the volume of left atrium indexed by BSA was similar in both groups. LVH was observed in 65 (82%) patients. The incidence of severe LVH as well as concentric and eccentric LVH were similar in both groups. There was no significant difference between groups in Doppler parameters assessing the diastolic function of LV. The mean (SD) value of average GLS for all patients was -19.3%
(-3.2%), however, Group 1 had significantly higher mean value of mean GLS ($P<0.005$).

The results from CART revealed the cut-off point for the HD duration as 27.5 months is related to GLS $\geq -18\%$ (Figure 1).

The similar CART analysis with time after KTx revealed the cut-off point for this period as 152.5 months is related to GLS $\geq -18\%$ (Figure 2).

Ten patients who did not undergo HD before KTx had a normal value of GLS (Figure 1). The mean (SD) time after KTx in these patients was 95.3 (51.8) months whereas the mean (SD) eGFR was 41.3 (9.1) mL/min/1.73m$^2$

The results of multivariate stepwise logistic regression analysis for variables related to impaired GLS were shown in Table 4. Only the two assessed variables such as the duration of HD longer than 27.5 months, and eGFR higher than 60mL/min/1.73m$^2$ were related to GLS $\geq -18\%$ but the period after KTx longer than 152.5 months had the only trend to relation with GLS $\geq -18\%$.

**Discussion**

This study demonstrates that impaired GLS occurs in nearly 40% of KTx patients with preserved LVEF. The GLS impairment is related to the presence and duration of HD before KTx and kidney allograft function assessed by eGFR.

In our study, the majority of KTx patients had severe LVH. The severity of LVH did not correlate with the impaired GLS. LVMI was comparable in patients with or without impaired GLS.

Impaired GLS in CKD patients is an indicator of decreased LV contractility probably due to LV remodelling with increased accumulation of extracellular matrix and myocardial fibrosis. Recently, Sandal et al. found that congestive heart failure, but not the coronary artery disease, is the most common cause of cardiovascular events following KTx [9]. That finding confirms earlier outcomes of studies based on the United States Renal Data System conducted on thousands of patients [10]. The consensus was that the KTx is a state of “accelerated heart failure” rather than “accelerated atherosclerosis”.

The advantages of LV GLS over LVEF in the assessment of LV systolic function have been demonstrated in several studies in general population as well as cardiac disease and in CKD patients.
Previously reported prevalence of impairment of LV GLS in CKD patients with preserved LVEF was between 17 and 60%. The differences could be attributed to the selection of CKD patients for the studies (with various stages of CKD from 1 to 5, involving patients with cardiac morbidity e.g. history of myocardial infarction, CABG, atrial fibrillation, heart failure), and various cut-off values of GLS (-18 to -15%). Panoulas et al. in their study carried out on 106 CKD patients with LVEF≥55% and no history of cardiovascular disease or its symptoms, defined the GLS impairment as values greater than -16% [11]. The incidence of impaired GLS was dependent on the CKD stage and was only 3.4% in stage 1 or 2, 39.5% in stage 3, and 25.6% in stage 4 and 5. While the greatest LVMI was observed in patient 4-5 stage. In patients with impaired GLS, the authors observed an increased rate of adverse cardiovascular events (including general mortality, coronary artery disease, and length of hospitalization for heart failure) during follow-up of 30.7±11.7 months. They also suggested that impaired GLS may be a result of microvascular ischemia and myocardial fibrosis as the studied group did not have an evident disease in epicardial arteries, which had been confirmed by elective angiography. Hensen et al. showed LV systolic dysfunction defined by LV GLS ≤15.2% in 32% of predialysis and dialysis patients with LV ejection fraction ≥50% [12]. Ravera et al. observed GLS impairment (defining the cut-off of GLS impairment value on less than -18%) in 55% of CKD patients in stage 2-4 and in 60% of dialysed patients [13]. In kidney transplant patients, according to various studies the value of GLS was -17 to -19%, because CKD patients with lower GLS were less frequently transplanted. In Hensen study, only 8% patients with GLS < 10.6% were transplanted, while 32% with GLS 10.7-15.1%; 49% with GLS 15.2-17.8%, and 47% with GLS > 17.9% [14].

Many previous studies have shown that KTx has a beneficial effect on LV structure and function compared to dialysis. The studies based on a series of conventional echocardiograms showed an improvement in LVEF after KTx. Ravinder et al. showed improvement not only in LVEF but also in functional status of heart failure and increased survival rate after KTx [15]. Paoletti et al. in their study also proved that the LVH regression following KTx was a predictor of a better long-term clinical outcome [16]. However, the LVM regression has not been confirmed by CMR what may raise doubts about the reversibility of LVH in transplant patients [17].
It has not been established whether GLS ameliorates following KTx. There are only few studies presenting GLS in small groups of patients before and after KTx. A retrospective study of Hewing et al. showed improvement of LV global longitudinal peak systolic strain (GLPS) after KTx [18]. They evaluated a group of 31 CKD patients at mean age 44 years before and 13-32 (mean 19) months after successful KTx and found significant improvement in LV systolic function assessed by GLS (-18.4%±2.8% vs -19.4%±2.3%). Concomitantly, reduction of LV end-diastolic septal and posterior wall thickness and LVMI were observed. In that study 8 patients (25%) had preemptive KTx. The analysis of LV systolic function assessed by speckle-tracking echocardiography was reported in pediatric patients with stage 3-5 CKD, during dialysis treatment, and after KTx. The study revealed higher impairment of GLS in patients on dialysis than in stage 3-5 CKD and in patients after KTx. One year after KTx, children presented impairment of GLS similar to stage 3-5 CKD, suggesting improvement of LV function. However, it cannot be determined whether impaired systolic contractility assessed by GLS did improve after KTx (in comparison to dialysis children) because 42% of patients (18 out of 42) had preemptive KTx [19].

Contrary to these studies, Gong et al. presented greater impairment of GLS in kidney transplant patients. They conducted a prospective study in 39 patients (mean age 47 years) who underwent KTx. LV GLS was assessed by cardiovascular magnetic resonance imaging (CMRI) at baseline and at 12 months after KTx. Kidney transplant patients had decline in GLS over 12 months (-15.9%±3% vs -14.9%±3%) post transplantation, although LVEF and LV volumes improved, pointing to a reduction of LVH [20].

We found a significant correlation between the impairment of GLS and duration of HD before KTx. Long HD time (> 27,5 months) increased the incidence of impaired GLS suggesting that the structural abnormalities of myocardium progress on HD and fail to subside after KTx. In contrast, ten patients with preemptive KTx had normal GLS value, although they were mean 95 months after transplantation and had lower eGFR (41.3 mL/min/1,73m²). Our study for the first time showed the relation of subclinical LV systolic dysfunction with a duration of HD before KTx. Relationship between LVH and duration of HD was reported. Foley et al. showed that after 18 months of dialysis 62% of the
patients had increased LV mass volume index and 49% of them developed overt LV failure [21]. Many studies showed, that long time on HD before transplantation is associated with decreased patient and kidney allograft survival. Meier-Krieshe study, including 73,103 patients with the United States Renal Transplant Scientific Registry, evidenced that longer time on HD prior to KTx compared to preemptive KTx was a significant risk factor for the death of patient with a functioning graft (p<0.001). Increase in mortality risk after KTx was 41% for patients on HD for 24-36 months and 72% for patients dialyzed over 48 months relative to preemptive transplantation [22]. Recently, Jay et al. reassessed preemptive KTx using data of 141,254 transplant recipients from United Network of Organ Sharing, who were transplanted between 2003 and 2012. Their retrospective study confirmed that preemptive KTx as well as short time of dialysis < 1 year prior to transplantation in recipients of living kidney donor were associated with higher 5-year patient survival. During the last 2 decades the percentage of patients with preemptive KTx raised from 10.9% to 17% [23].

During HD the patients are exposed to multiple factors such pressure and volume overload, activation of renin-angiotensin system chronic inflammation, hiperhomocysteinemia, advanced glycosylation end products, anemia, endothelial dysfunction, oxidative stress, arteriovenous access. The cumulative effect of these factors leads to cardiac and vascular damage. Dialysis treatment compared to preemptive KTx was associated with increased stiffness and reduced vascular compliance. Yet, the important role of FGF23 should emphasized, which serum level increase over 400 times in hemodialysed patients. FGF23 levels are associated with LVH, and myocardial fibrosis, and increased cardiovascular mortality [24].

In our study value of eGFR > 60mL/min/1.73m² was a protective factor for the development of subclinical LV systolic dysfunction. The GLS relationship to eGFR in kidney transplant recipients has not been studied so far. However, in CKD patients before transplantation LV systolic function progressed with falling eGFR and was the worst during dialysis. The study of Park et al. proved that the decrease of eGFR >60 mL/ min/1.73 m² correlate with impaired LV function on follow-up echocardiography [25]. Normal renal function after transplantation reduces the risk of cardiovascular
mortality in kidney transplant recipients but injured renal function is a strong risk factor for cardiovascular events and mortality. KTx restores normal renal function (eGFR >90 mL/min/1.73 m²) only in 5% of recipients. The remaining recipients present stage 3A (30%), 3B (18%) and stage 4 (5%) of CKD [26]. Weiner et al. in FAVORIT study showed that decrease of eGFR below 45 mL/min/1.73 m² is associated with cardiovascular disease and mortality, while increase of eGFR by each 5 mL/min/1.73 m² above 45mL/min/1.73 m², is associated with 15% reduction in CVD and mortality [27].

We also observed that impaired GLS borderline depend on the time elapsed after KTx. Patients who were transplanted more than 152,5 months had higher occurrence of impaired GLS however in stepwise logistic regression analysis it was not statistically significant.

Ten patients in our study with preemptive KTx had normal GLS, although they were mean 95 months after transplantation and had lower eGFR (41.3 mL/min/1.73 m²) This outcome emphasises the most harmful role of HD on systolic function on LV

Conclusions

In total, 39% of kidney transplant patients with preserved LVEF and no history of ischemic heart diseases had reduced LV contractility defined as impaired GLS. The main risk factor of GLS impairment was a long period of HD (>27,5months) prior to the transplantation procedure. Good renal graft function with eGFR>60mL/min/1.73m² after KTX occurred to be a protective factor for GLS impairment.

Abbreviations

A late diastolic mitral inflow

a peak late diastolic filling velocity

BMI body mass index

BSA body surface area

CART Classification and Regression Tree Analysis

CKD chronic kidney diseases

DM diabetes mellitus
E  early diastolic mitral inflow
e  early diastolic filling velocity
eGRF  estimated glomerular filtration rate
GLS  global longitudinal strain
HD  haemodialysis
HTN  hypertension
IVDd  intraventricular end-diastolic diameter posterior end-diastolic wall thickness diameter LAd  left atrium diameter
LV  left ventricle
LVEDd  left ventricle end-diastolic diameter
LVEF  left ventricle ejection fraction
LVH  left ventricle hypertrophy
LVM  left ventricle mass
LVMI  left ventricle mass index
KTx  kidney transplantation
PWTd  posterior end-diastolic wall thickness diameter
RWT  relative wall thickness
SD  standard deviation
STE  speckle-tracking echocardiography

Declarations

Ethics approval and consent to participate
The study is retrospective and has been approved by the Ethics Committee of Wroclaw Medical University – The statement No 530/2019

The consent for publication
Not applicable

Availability of data and materials
The datasets used and analysed during the current study are available from the corresponding author
on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

OM performed the echocardiography examinations and was a major contributor in writing the manuscript. OM, GA, SM contributed to the design of the research. SM BM, LK KM analyzed and interpreted the patients` data regarding haemodialysis and transplantation. All the authors were involved in data collection. OM, ZD performed statistical analysis. All authors read and approved the final version of the manuscript.

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Tables

Table 1. The demographic and clinical characteristics.
|                           | Group 1 (n=31)          | Group 2 (n=48)          | P    |
|---------------------------|-------------------------|-------------------------|------|
| Age                       | 60.9±10.4               | 59.9±12.3               | 0.36 |
| Gender female             | 14 (45.2%)              | 28 (58.5%)              | 0.25 |
| BSA, m²                   | 1.91±0.19               | 1.79±0.22               | 0.03 |
| BMI, m²/kg                | 27.12±3.90             | 25.48±4.39             | 0.09 |
| HD, months                | 31.8±29.2               | 18.4±17.2               | 0.01 |
| HD>27,5 months            | 15 (48.4%)              | 9 (18.8%)               | 0.005|
| KTx, months               | 127.2±69.5              | 115.7±73.19             | 0.48 |
| KTx>152,5 months          | 13 (41.9%)              | 10 (20.8%)              | 0.22 |
| No HD before KTx          | 0 (0%)                  | 10 (20.8%)              |      |
| DM                        | 9 (29.0%)               | 12 (25.0%)              | 0.75 |
| HTN                       | 27 (87.1%)              | 45 (93.7%)              | 0.76 |

Data are shown as number (percentage), mean ± standard deviation. Group 1 represent patients with GLS≥-18%, whereas Group 2 - GLS<18%.

BMI - body mass index, BSA - body surface area, DM - diabetes mellitus, HD - haemodialysis, HTN - hypertension, KTx - kidney transplantation

Table 2. The laboratory results.
|                           | Group 1 (n=31) | Group 2 (n=48) | P   |
|---------------------------|----------------|----------------|-----|
| **Total cholesterol, mg%**| 214.8±59.9     | 219.1±56.6     | 0.74|
| **HDL, mg%**              | 54.2±11.9      | 59.3±18.8      | 0.18|
| **LDL, mg%**              | 129.6±41.4     | 127.6±40.9     | 0.83|
| **Triglycerides, mg%**    | 174.29±103.80  | 167±4±69.6     | 0.72|
| **Serum creatinine, mg%** | 1.73±0.85      | 1.63±0.72      | 0.56|
| **eGFR, mL/min/1.73 m²**  | 42.94±15.77    | 45.52±16.36    | 0.49|
| **eGFR mL/min/1.73 m²**   | 2osoby6,5      | 9 18,8         | 0.23|
| **Uric Acid, mg%**        | 6.92±1.42      | 6.91±1.62      | 0.98|

Data are shown as number (percentage), mean ± standard deviation. Group 1 represent patients with
GLS≥-18%, whereas Group 2 - GLS<-18%.
eGFR - estimated glomerular filtration rate, HDL - high-density lipoprotein cholesterol, LDL - low-density lipoprotein cholesterol.

Table 3. The echocardiographic parameters.

|                          | Group 1 (n=31) | Group 2 (n=48) | P   |
|--------------------------|----------------|----------------|-----|
| LVDd, mm                 | 51.0± 5.5      | 48.5±5.7       | 0.052 |
| IVDd, mm                 | 13.6±1.9       | 13.2±3.1       | 0.56 |
| PWd, mm                  | 11.1±1.4.3     | 10.9±1.2       | 0.42 |
| LAd, mm                  | 43.5±4.2       | 41.2±3.0       | 0.006 |
| LAV                      | 72.3 ±21.2     | 60.8±16.5      | 0.01 |
| LAVI                     | 38.1± 11.8     | 34.0± 7.5      | 0.07 |
| EF, %                    | 59.8±5.5       | 61.9±4.4.      | 0.07 |
| LVMI in women,g/m2       | 121.1±34.7     | 123.0 ± 38.6   | 0.88 |
| LVMI in men g/m2         | 145.4 ± 31.0   | 132.6 ± 31.1   | 0.22 |
| Without LVH              | 5(16.1%)       | 9(18.8%)       | 0.77 |
| Severe LVH               | 14(45.2%)      | 17(35.4)       | 0.39 |
| RWT                      | 0.44 ± 0.06    | 0.45 ± 0.07    | 0.33 |
| Excentric LVH            | 6 (19.4%)      | 7 (14.6%)      | 0.58 |
| Concentric LVH           | 15 (48.4%)     | 23 (47.9%)     | 0.97 |
| E, cm/s                  | 67.78±21.9     | 66.2±14.7      | 0.71 |
| A, cm/s                  | 79.8±20.4      | 84.2±16.8      | 0.30 |
| DT, ms                   | 233.7±66.7     | 221.5±56.8     | 0.38 |
| e lateral, cm/s          | 7.4±1.6        | 7.8±2.1        | 0.31 |
| a lateral, cm/s          | 9.4±2.6        | 10.5±2.6       | 0.06 |
| e septal, cm/s           | 6.2±1.6        | 6.4±1.9        | 0.60 |
| a septal, cm/s           | 9.5±3.2        | 9.5±2.1        | 0.92 |
| E/e avr>10               | 23 (74.2)      | 37 (77.1%)     | 0.77 |
| E/e avr<10               | 11 (35.5%)     | 19 (39.6%)     | 0.71 |
| E/e avr>14               | 3 (9.7%)       | 2.4 (2%)       | 0.33 |
| GLS average %            | -16.3±1.9      | -21.3±2.1      | <0.005 |

Data are shown as number (percentage), mean ± standard deviation. Group 1 represent patients with GLS≥-18%, whereas Group 2 - GLS<-18%.

GLS - global longitudinal strain, LVH - left ventricular hypertrophy, LVDd - left ventricle end-diastolic diameter, LVDs - left ventricle end-systolic diameter, EF - ejection fraction, LAd - left atrium diameter, LVM - left ventricle mass, LWMI - indexed left ventricle mass, E - early mitral flow, A - atrial mitral
flow, e - early mitral annulus flow, a - atrial annulus mitral flow, DT - deceleration time.

Table 4. The results of the stepwise logistic regression analysis for independent variables related to GLS≥-18%.

|                                | OR   | -95%CI        | P    |
|--------------------------------|------|---------------|------|
| Duration of HD before KTx > 27.5 months | 4.58 | 1.46-14.38    | 0.01 |
| eGFR > 60ml/min/1.73 m²          | 0.155| 0.024-0.967   | 0.04 |
| Duration of time after KTx       | 2.43 | 0.80-7.43     | 0.12 |

eGFR - estimated glomerular filtration rate, HD - haemodialysis, KTx - kidney transplantation.

Figures
**Figure 1.** The Classification and Regression Trees using the presence of haemodialysis, and duration of haemodialysis prior to kidney transplantation determining the incidence of GLS≥-18%.

The global CV =0.60 sd CV cost 0.05

The number in the upper right corner depicts the prevalence in the given subgroup of GLS<-18% (0) or GLS≥18% (1).

CART - Classification and Regression Trees, GLS - global longitudinal strain, HD - haemodialysis, MO – months.

**Figure 1**

The Classification and Regression Trees using the presence of haemodialysis, and duration of haemodialysis prior to kidney transplantation determining the incidence of GLS≥-18%.

The global CV =0.60 sd CV cost 0.05 The number in the upper right corner depicts the prevalence in the given subgroup of GLS<-18% (0) or GLS≥18% (1). CART - Classification and Regression Trees, GLS - global longitudinal strain, HD - haemodialysis, MO – months.
The global CV = 0.57 sd CV cost 0.06.

The number in the upper right corner depicts prevalence in the given subgroup of GLS<18% (0) or GLS≥18% (1).

CART - Classification and Regression Trees, GLS - global longitudinal strain, KTx - kidney transplantation, MO - months

Figure 2

The Classification and Regression Trees using the presence of haemodialysis and duration of time after kidney transplantation determining the occurrence of GLS≥-18%. The global CV = 0.57 sd CV cost 0.06. The number in the upper right corner depicts prevalence in the given subgroup of GLS<18% (0) or GLS≥-18% (1). CART - Classification and Regression Trees.
Trees, GLS - global longitudinal strain, KTx - kidney transplantation, MO - months