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# Differences in Cardiovascular Remodeling in Kidney Transplant Recipients and Peritoneal Dialysis Patients

**Rebić Damir [a*], Hamzić-Mehmedbašić Aida [a], Džubur Alen [b], Granov Nermir [b] and Hasanspahić Senad [a]**

*[a] Nephrology Clinic, Clinical Center University of Sarajevo, Sarajevo, Bosnia and Herzegovina.*
*[b] Cardiology Clinic, Clinical Center University of Sarajevo, Sarajevo, Bosnia and Herzegovina.*

***Authors' contributions***

*This work was carried out in collaboration among all authors. Author RD designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors HMA and DA managed the analyses of the study. Authors GN and HS managed the literature searches. All authors read and approved the final manuscript.*

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## ABSTRACT

**Aims:** Cardiovascular disease (CVD) is the leading cause of death in dialysis patients as well as in kidney transplant recipients (KTx). Left ventricular hypertrophy (LVH) starts early during the course of chronic kidney disease and is a strong predictor of CVD. We hypothesised that kidney transplant is significantly associated with improvement in cardiovascular reserve. We conducted a prospective study to compare changes in CV before and after kidney transplantation in patients with ESRD who received KTx to control individuals who received PD but did not receive a KTx.
**Study Design:** A Case-Control Study.

**Corresponding author: E-mail: damir.rebic@gmail.com;*

**Place and Duration of Study:** Clinic for nephrology Clinical Center University of Sarajevo, Bosnia and Herzegovina.
**Methodology:** In this case-control study, we included 50 KTx from the Kidney Transplant Outpatient Clinic for nephrology Clinical Center. For each 50 KTx, PD outpatients matched for gender and age were recruited. All patients underwent transthoracic echocardiography, and LV (left ventricular) mass (LVM), LV mass index (LVMi), and indices of cardiac function were measured. In the small subgroup of 18 KTx, we retrospectively assessed and compared the LVMI measurements, during dialysis and the post-transplant period.
**Results:** The prevalence of LVH was 24% in KTx patients and 72% in PD patients (*NS*). KTx had significantly lower LVM, LVMi levels, E/A ratio, FS, and LA diameter compared with the PD group, while the EF and other echocardiographic parameters did not differ. In the subgroup of 18 KTx, LVMi levels after transplantation were significantly lower than dialysis LVMi levels.
**Conclusion:** LVH is the most frequent cardiac abnormality at the time of kidney transplantation. After KTx, the reduction of LVH and diastolic dysfunction was significant. CV remodelling after successful KTx is related to better kidney function, and can explain better outcomes for patients with kidney transplants over patients on long-term dialysis.

*Keywords: Kidney transplantation; peritoneal dialysis; cardiovascular remodeling; echocardiographic measurements.*

## 1. INTRODUCTION

"Cardiovascular disease (CVD) is highly prevalent and a leading cause of death among patients undergoing renal replacement therapy (RRT)" [1]. "Left ventricular hypertrophy (LVH) and systolic and diastolic dysfunction are well-recognized indicators of worse cardiovascular outcomes in dialysis patients. In advanced chronic kidney disease (CKD), the myocardium is exposed to complex metabolic stressors that result from uremia-related inflammation, oxidative stress, renin-angiotensin-aldosterone system activation, calcitriol and klotho deficiency, increased fibroblast growth factor (FGF) 23, and changes in mineral metabolism" [2]. "This exposure leads to myocyte hypertrophy, reduced myocardial capillarization, and nonvascularized interstitial fibrosis, as well as arteriosclerosis and arterial stiffening" [3]. "Together, these ultrastructural changes reduce pump efficiency and increase cardiac energy expenditure and myocardial oxygen consumption. LVH starts early in the course of CKD and is inversely correlated with kidney function" [4]. At the beginning of dialysis, 75% of patients with end-stage renal disease (ESRD) have LVH, primarily because of hypertension, volume expansion, and anaemia. Kidney transplantation is the optimal treatment for ESRD and is associated with reduced cardiovascular morbidity and improved quality of life and survival [5]. Some echocardiographic studies have reported reduced left ventricular mass and improved left ventricular ejection fraction (LVEF), but these findings have been inconsistent. In fact, serial cardiac magnetic resonance imaging has failed to detect a significant reduction in left ventricular mass following kidney transplantation. In a number of studies, successful kidney transplantation has been associated with significant echocardiographic regression of LVH in KTx (kidney transplant) [5,6], while other studies have shown no positive effect. The expansion and variation of intravascular volume in dialysis patients compared with KTx have been suggested as one of the reasons for the contradictory result [7,8].

"The mortality rate in patients with ESRD is much higher than in the general population, despite advances in dialysis treatment. Cardiovascular structure and functional abnormalities, such as LVH, left ventricular (LV) systolic and diastolic dysfunction, accelerated atherosclerosis, arrhythmias, and coronary artery calcification, contribute to high cardiovascular mortality in patients with ESRD" [9].

We hypothesised that a kidney transplant is significantly associated with improvement in cardiovascular reserve. We conducted a prospective study to compare changes in cardiovascular parameters before and after kidney transplant in ESRD patients who received kidney transplants to control individuals who received peritoneal dialysis (PD) but did not receive a kidney transplant.

## 2. MATERIALS AND METHODS

### 2.1 Patients

In this case-control study, we included 50 KTx from the KTx Outpatient Clinic of nephrology Clinical Center University of Sarajevo, Bosnia and Herzegovina. For each 50 KTx, chronic PD outpatients matched for gender and age were recruited. "Exclusion criteria for all patients were any cardiovascular event (defined as myocardial infarction, acute ischemic heart disease, stroke, and peripheral vascular disease,) occurring within a period of 6 months prior to study entry, severe heart failure at NYHA stages III and IV, any moderate or severe valvular heart disease, the presence of clinical infection, and active malignancy. Moreover, in a subgroup of eighteen KTx the LVM (left ventricular mass) index (LVMi) measurements were retrospectively assessed at two different time points, the first during the predialysis period and the second during dialysis treatment, and compared with the LVMi estimation during the posttransplant period. All KTx had a functioning allograft for at least six months and received calcineurin inhibitor-based (tacrolimus or cyclosporine A) immunosuppression" [10]. The local hospital's Ethical Committee approved the study, and patients participated in it after providing informed consent.

### 2.2 Methods

At study entry, all patients underwent a review of their medical history and a clinical examination. Additionally, demographic characteristics, co-morbidities (CVD, diabetes mellitus, and hypertension), medications, and blood pressure (BP) were assessed. Hypertension was defined as systolic BP >140 mm Hg and diastolic BP >90 mm Hg, or the current use of antihypertensive medication. A full biochemical screen was performed, urine protein was measured in a 24-hour urine collection, and eGFR (mL/min/1.73 m2) was calculated by the CKD-MDRD formula [11].

### 2.3 Echocardiographic Data

"Comprehensive echocardiographic measure-ments were performed using an ultrasound machine (Toshiba 270SSA) with a 3.75-MHz sector probe by a single experienced cardiologist blinded to clinical information on patients at baseline and at the end of the study period. All images were obtained with standard techniques using M-mode, two-dimensional, and Doppler measurements in accordance with the American Society of Echocardiography guidelines" [12]. "The LV mass was calculated using the modified formula proposed by Devereux et al. [13]. Echocardiographic evidence of LVH was defined as the LV mass index divided by a body surface area of >115 $g/m^2$ in men and >95 $g/m^2$ in women. LV systolic function was assessed by calculation of the ejection fraction (EF) using a modified Simpson's method" [14], while fractional shortening (FS) was calculated according to the formula described by Lang et al. [15]. "LV systolic weakness was defined as EF <50% and FS <30%. Pulsed Doppler echocardiography was used to evaluate transmitral LV filling velocities at the tips of the mitral valve. The peak early-diastolic flow velocity (E) and the peak late-diastolic flow velocity (A), shown as the E/A ratio, were measured by analysing the transmitral flow. LV diastolic dysfunction was defined as E/A ≤1" [16].

### 2.4 Statistical Analysis

"Data were presented as mean and standard deviation (for normally distributed data), median and interquartile range (for not-normally distributed data), or as absolute count and frequency in percent (for binary variables). The chi-squared or Fisher exact test was used for categorical variables, whereas comparisons of continuous variables among the two groups of patients were analysed using the student's t-test. Multivariate linear regression analysis was performed to determine the factors that were independently associated with LVMi levels in both groups of patients. Statistical significance was defined as p values less than.05. All analyses were performed using SPSS 21.0 (SPSS, Chicago, IL)" [10].

## 3. RESULTS

The baseline characteristics of the 50 KTx and 50 PD patients are summarised in Table 1. According to the study design, the two patient groups were matched for sex and age. There were no differences in co-morbidities (arterial hypertension, diabetes mellitus, and cardiovascular disease) or blood pressure levels between the two groups (Table 1). Regarding laboratory parameters, there were no significant differences in levels of haemoglobin (Hb), cholesterol, and phosphorus, between the study groups. Twenty-four hours of proteinuria content

was significantly lower in the KTx group (P .002) and serum calcium was significantly higher compared to the PD group, as expected (P .05) (Table 1).

In terms of medication, RAAS inhibitors and diuretics were used by a significantly higher proportion of PD patients than in the KTx group (P =.033 and.042, respectively).

In the KTx group of all patients, 38 of them (76%) showed normal echocardiographic findings, and 24% of patients had echocardiographic signs of left ventricular hypertrophy. Among patients with LVH, 47% (14 pts) of them had concentric and 20% had eccentric LV hypertrophy. After at least 12 months of PD treatment, 72% of patients developed LVH.

In the study, both groups had normal average systolic and diastolic heart function, although the PD group had borderline echocardiographic parameters in terms of LV systolic and diastolic dysfunction. KTx group remained with a normal LV mass index, unlike the PD group, as shown in Table 2.

Examining predictors of LVMi in the KTx group using the model of logistic regression analysis, it was found that diastolic blood pressure and CRP were independent positive predictors, and urine proteinuria per day was an independent negative predictor of LVMi (Table 3).

**Table 1. Baseline data of KTx and PD patients**

| Parameters | KTx (50 pts.) | PD (50 pts.) | P |
|---|---|---|---|
| Sex (M/F) (n) | 27/23 | 31/19 | NS |
| Age | 49.5 ± 9.8 | 53.6 ± 11 | NS |
| CVD (n, %) | 11 (22%) | 16 (32%) | NS |
| DM (n, %) | 6 (12%) | 19 (38%) | NS |
| Hypertension (n, %) | 32 (64%) | 41 (82%) | NS |
| eGFR-MDRD (mL/min/1.73 $m^2$) | 58.5±16.3 | 16.1±3.12 | .05 |
| Proteinuria (mg/24h) | 239 (66-424) | 403 (256-2296) | .002 |
| Serum albumin (g/L) | 32±3.3 | 26±8.4 | .04 |
| Systolic BP (mmHg) | 136 ± 17 | 138±21 | NS |
| Diastolic BP (mmHg) | 82±8 | 84±10 | NS |
| Hb (g/L) | 133 ±15 | 107 ±27 | NS |
| Ca (mmol/L) | 2,31±0.5 | 2.19±0.8 | .05 |
| PO4 (mmol/L) | 1.67±0.3 | 1.77±0.7 | NS |
| PTH (pg/ml) | 98 (56-137) | 148 (107-262) | .05 |
| Cholesterol mmol/L | 4.73±0.73 | 7.05±0.94 | NS |
| Trygliceride mmol/L | 1.51±0.40 | 2.84±0.34 | .032 |
| LDL mmol/L | 2.90±0.36 | 4.45±0.86 | .04 |
| CRP (mg/L) | 1.5 (0.8- 3.3) | 5.9 (2.3-11) | <.001 |
| RAAS inhibitor (n, %) | 17 (34%) | 42 (84%) | .033 |
| Diuretics (n, %) | 9 (18%) | 24 (48%) | .042 |
| β-blockers (n, %) | 34 (94%) | 19 (26%) | .045 |
| Ca channel blockers (n, %) | 20 (68%) | 31 (62%) | NS |
| Statins (n, %) | 22 (44%) | 24 (48%) | NS |
| Erythropoietin (n, %) | 9 (18%) | 26 (52%) | .023 |
| Duration KTx (years) (IQR) | 2.9 (1.2-4.4) | - | - |
| Duration PD (years) (IQR) | - | 2.1 (1.0-4.8) | - |

*Data are expressed as the mean ± SD, number (percentage) or median (range); NS= no significant; IQR=interquartile range*

***Legend:*** *KTx-kidney transplant; PD-peritoneal dialysis; CVD-cardiovascular disease; DM-diabetes mellitus; eGFR-MDRD-estimated glomerular filtration rate - Modification of Diet in Renal Disease; BP-blood pressure; PO4-phosphorous; PTH-parathyroid hormone; LDL-low-density lipoprotein; CRP-C-reactive protein; RAAS-renin-angiotensin-aldosterone system; Ca-calcium*

**Table 2. Echocardiographic Measurements in KTx and PD patients**

| Parameters | KTx | PD | *P* |
|---|---|---|---|
| EF (%) | 68.1±8.4 | 56.9±10.0 | NS |
| FS (%) | 37.8±7.1 | 30.8±4.1 | <.001 |
| E/A ratio | 1.1±0.1 | 1.0±0.1 | .042 |
| LVEDD (mm) | 49.6±6.5 | 52.9±3.6 | <.001 |
| LVESD (mm) | 35.0±3.5 | 34.3±3.9 | NS |
| LVMi ($g/m^2$) | 103.2±32.1 | 140.2±37.8 | <.001 |
| LVM (gr) | 190.7±68.2 | 243.8±40.8 | <.001 |
| LV volume ($mL/m^2$) | 83.8±21.7 | 90.2±24.5 | <.001 |
| LA Diameter (mm) | 40.5±6.7 | 43.3±5.7 | .38 |
| LVH (n, %) | 12(24) | 36(72) | NS |

*Data are expressed as the mean ± SD, number (percentage) or median (range); NS= no significant*
***Legend:*** *EF-Ejection fraction; FS-Fractional shortening; LVM Left ventricle mass; LVMi Left ventricle mass index; LVEDD - LV end-diastolic diameter; LVESD - LV end systolic diameter; LA- Left atrium*

**Table 3. Independent Factors Associated With LVMi in KTx**

| Variables | β | Sig | CI 95% (Lower-Upper Bound) |
|---|---|---|---|
| Diastolic BP | 0.364 | 0.012 | 0.353- 2.596 |
| Proteinuria/day | -0.102 | 1.001 | 1.641-27.27 |
| CRP | 0.453 | 0.171 | 1.124-2.200 |

***Legend:*** *LVMi-left ventricular mass index; KTx-kidney transplant; BP-blood pressure; CRP- C-reactive protein*

**Table 4. Independent factors associated with LVMI in PD**

| Variables | β | Sig | CI 95% (Lower-Upper Bound) |
|---|---|---|---|
| Diuresis/day | -0.002 | 0.001 | 0.217**-** 0.612 |
| Cholesterol | 2.395 | 0.725 | 2.647-45.433 |
| LDL | 3.604 | 1.011 | 5.066-26.359 |
| Hemoglobin | -0.418 | 0.155 | 0.485-0.893 |
| Serum albumin | -1.111 | 0.459 | 0.134-0.809 |

***Legend:*** *L LVMi-left ventricular mass index; PD-peritoneal dialysis; LDL-low-density lipoprotein*

Independent negative predictors of the LVMi in the PD group were 24-hour diuresis (daily collection of urine), hemoglobin, and serum albumin level, and total cholesterol and low-density lipoprotein were independent positive predictors of left ventricular remodelling (Table 4).

## 4. DISCUSSION

This study provides an integrated assessment of cardiac functional and morphologic changes in PD and kidney transplant patients. "Cardiovascular disease accounts for half of the deaths in patients treated with RRT, whereas mortality from CV causes is far higher than in the general population" [2,16].

"The influence of uremia and dialysis on CV structure and function was investigated, as detailed by studies on patients with CKD" [17,18].

"In contrast to the limited prognostic value of single surrogate factors of most established clinicopathologic factors (such as age, arterial hypertension, LVMi, and LVEF), measures of CV reserve have been shown to be independently associated with survival in patients undergoing peritoneal dialysis and after adjusting for known comorbid factors" [19].

"Two parallel processes are involved in the development of cardiovascular disease in dialysis patients. One of the first processes involves cardiac remodeling, including LVH and

LV dysfunction, as a response to mechanical or hemodynamic load. The following process involves vascular remodeling, including atherosclerosis, arteriosclerosis, and vascular calcification. In recent times, cardiac structural and functional changes such as left ventricular hypertrophy and dysfunction, and vascular alterations, e.g., arterial stiffness, increased intima-media thickness (IMT), and coronary calcification, have been accepted as early markers of cardiovascular remodelling" [20]. "LV diastolic dysfunction is frequently observed in dialysis patients and results from LVH, cardiac fibrosis, and impaired cardiac relaxation. In addition, the presence of LVH and LV systolic dysfunction are well-known risk factors for CV mortality in the dialysis population" [21].

Considering the high risk, our data may emphasise the importance of early recognition of LV structure and function changes and may have significant therapeutic implications in the treatment of both dialysis and transplant patients. In our study, KTx improvements were significant even after adjusting for age, body mass index, sex, diabetes, cardiovascular comorbidity, use of RAAS inhibitors or other antihypertensive drugs, and anemia. Despite the fact that symptoms of chronic uremia and volume load were significantly less two months after transplantation, true improvement was noticeable twelve months after transplantation, while no such improvements were noted in the PD group. Of course, such results are consistent with incomplete normalisation of kidney function after transplantation. Based on these data, it can be suggested that the structural and molecular changes in the cardiovascular system caused by uremia can be partially regressed, which can result in a significant and visible cardiovascular functional improvement.

A series of prospective studies have evaluated pretransplant and posttransplant kidney ultrasound left ventricular dimensions and function [22,23].

Some authors have reported a significant regression of LVMI (but no change in systolic function) in kidney transplant recipients after the first post-transplant year. On the other hand, there was no change in LVMI and a significant improvement in systolic function at an average of three years after transplantation among the subjects [24]. In patients with a pre-transplantation ejection fraction of less than 50%, the authors reported an increase in systolic function 1 and 12 months after kidney transplantation. These studies are limited by the small number of patients studied, the lack of non-transplant controls, and the lack of data on the timing of echocardiography with dialysis, given the potential for confounding by vascular volume or dialysis-induced myocardial stunning [25].

Factors that may be involved in LVH and fibrosis in chronic kidney disease but also in ESRD patients are elevated systolic and diastolic blood pressure, volume overload, and anaemia due to erythropoietin deficiency [26]. Interestingly, in our group of patients with PD, apart from traditional risk factors, 24-hour diuresis remained an independent factor associated with the level of LVMI. Unlike hemodialysis, which is associated with marked fluctuations in body water content due to intermittent dialysis treatment, PD is characterised by an almost steady state, which likely has a major impact on left ventricular function. In addition, data from this study show that residual renal function is lower in ESRD without dialysis compared to patients undergoing peritoneal dialysis. Wang [27] found that residual renal function may play a significant role in limiting the increase in cardiovascular remodelling by improving total Kt/V urea and removing toxins characteristic of the chronic uremic state.

Such results assume the existence of some non-dialyzable uremic toxins that cannot be removed by dialysis treatment and are significant in accelerating pathological cardiovascular changes in this population.

"After kidney transplantation, parameters that are significantly associated with regression of left ventricular mass are allograft function, left ventricular mass before transplantation, successful control of blood pressure, and adequate correction of anaemia" [24]. In our KTx group, multivariate analysis showed that independent factors associated with LVMI were diastolic blood pressure, but also 24-hour proteinuria, and acute inflammatory factor levels.

It is important to emphasise that our results did not reveal significant differences in terms of systolic and diastolic AT between the two observed groups of patients, while the KTx group still had a significantly lower protein excretion compared to the PD group. Proteinuria was found to be independently and significantly associated with left ventricular hypertrophy in

KTx patients but not in PD patients, according to our findings.

The most commonly used drugs in both groups were beta-blockers, statins, and erythropoietin, as well as immunosuppressive drugs for transplant patients.Using a cardioselective - blocker in ESRD patients with dilated cardiomyopathy reduced sudden cardiac death, whereas correction of anaemia with erythropoietin substitution had no significant beneficial effect on LVH [28]. Also, statin treatment reduced the risk of a major cardiac event in KTx, but its relationship with the regression of left ventricular mass was not investigated [29]. Based on the existing literature, we think that the significant difference in the left ventricular mass index between the two groups of patients in our study cannot be fully explained by differences in the level of proteinuria and the use of similar drugs. Decreased volume expansion after successful renal transplantation and good control of modifiable risk factors for LVH in our KTx could partly explain our results of better LV mass in this group compared with patients who underwent PD.

In a very small group of only 18 KTx, a reduction in LVMI was verified after kidney transplantation with a functioning allograft. Following our results, a significant number of other studies have shown significant cardiac changes after kidney transplantation. One study evaluated KTx patients before and 3 months after kidney transplantation and showed a significant improvement in systolic function and a decrease in the diameter of the heart cavities [30], while another study showed normalisation of fractional shortening in a group of patients with systolic dysfunction with a decrease in LVMi and LV diastolic volume [31]. “A recent study in KTx showed that more than 50% of patients experienced regression of LV mass after kidney transplantation” [30]. Also, in this small KTx group of ours, we found a reduction in LVMI compared to their ultrasound assessments before dialysis treatment. One possible explanation could be a higher level of eGFR after transplantation compared to eGFR before dialysis. Although we did not find a significant correlation between LVMI and eGFR at any time, other studies have shown that the severity of left ventricular structural remodelling is inversely proportional to residual renal function in patients on dialysis and in KTx [27]. Our finding is limited by the small number of patients and variability in echocardiographic measurements, but to our knowledge, this is one of the rarest studies that evaluated and compared serial measurements of LVM in the same patient population, starting with follow-up from the early period of peritoneal dialysis to the posttransplantation phase.

“Left ventricular hypertrophy is now considered a strong risk factor for increased cardiovascular mortality in all groups of patients with severe chronic kidney disease. Whether successful kidney transplantation can reverse LVH remains a debatable question” [10]. Finally, we found that KTx had significantly lower LVM compared with PD patients matched for age and residual renal function. Indeed, we have shown a partial regression of LVH after kidney transplantation. Although the small sample of patients limits our study, our data may imply that both LVM levels before ESRD and during dialysis may significantly influence LVM after transplantation. Prospective studies with a larger number of subjects are needed to assess the course of LVH at the start and throughout the progression of CKD.

## 5. CONCLUSION

Our study discovered that partial kidney function restoration by transplant was significantly associated with improved cardiovascular functional status without a significant change in ventricular structural morphologic features. The study seems to shed light on cardiovascular structural-functional dynamics and the relationship between the restoration of kidney function and cardiovascular physiologic results. Left ventricular hypertrophy is the most common cardiac structural change at the time of kidney transplantation. After transplantation, regression of LVH and better diastolic function were significant. Cardiovascular remodelling after kidney transplantation is associated with good allograft function, with correction of anaemia and control of mineral imbalance and parathyroid hormone secretion, and thus can explain the more favourable outcome for kidney transplant patients compared to patients on long-term dialysis.

## CONSENT AND ETHICAL APPROVAL

Informed consent was taken from all patients for the study participation which is performed with the approval of the Ethics committee, Clinical Center University of Sarajevo (04-229-10-2022).

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

## REFERENCES


1. Cozzolino M, Mangano M, Stucchi A, Ciceri P, Conte F, Galassi A. Cardiovascular disease in dialysis patients. Nephrol Dial Transplant. 2018;33(suppl_3): iii28-iii34.
   DOI: 10.1093/ndt/gfy174
2. Rangaswami J, McCullough PA. Heart Failure in End-Stage Kidney Disease: Pathophysiology, Diagnosis, and Therapeutic Strategies. Semin Nephrol. 2018;38(6):600-617.
   DOI: 10.1016/j.semnephrol.2018.08.005
3. Di Lullo L, House A, Gorini A, Santoboni A, Russo D, Ronco C. Chronic kidney disease and cardiovascular complications. Heart Fail Rev. 2015;20(3):259-72.
   DOI: 10.1007/s10741-014-9460-9
4. Alprecht-Quiroz P, Zúñiga-Pineda B, Lara-Terán JJ, Cáceres-Vinueza SV, Duarte-Vera YC. Síndrome cardiorrenal: aspectos clínicos y ecocardiográficos [Cardiorenal syndrome: Clinical and echocardiographic aspects]. Arch Cardiol Mex. Spanish. 2020;90(4):503-510.
   DOI: 10.24875/ACM.20000087
5. Reza N, De Feria A, Wang T, Chowns JL, Hoffman-Andrews L, Kim J, Hornsby N, Marzolf A, Atluri P, Herrmann HC, Owens AT. Left Ventricular Hypertrophy and Hypertrophic Cardiomyopathy in Adult Solid Organ Transplant Recipients. Transplant Direct. 2021;8(1):e1279.
   DOI: 10.1097/TXD.0000000000001279
6. Sreedharan S, Mathew A, Paul Z, Mathew N, Sundaram KR, Kurian G, Nair R. Changes in Left Ventricular Mass and Cardiovascular Risk Factors after Kidney Transplantation. Saudi J Kidney Dis Transpl. 2020;31(5):1006-1013.
   DOI: 10.4103/1319-2442.301165
7. Hashi AA, Ramesh Prasad GV, Connelly PW, Deva DP, Nash MM, Yuan W, Wald RM, Wald R, Gunaratnam L, Lok CE, Kim SJ, Karur GR, Connelly KA, Yan AT. Cardiac MRI assessment of the right ventricle pre-and post-kidney transplant. Int J Cardiovasc Imaging. 2021;37(5): 1757-1766.
   DOI: 10.1007/s10554-020-02153-4.
8. Malyszko J, Malyszko JS, Hryszko T, Brzosko S, Lebkowska U, Mysliwiec M. Renal transplant recipients with coronary artery disease exhibit impairment in fibrinolysis and structural changes in carotid arteries. Transpl Int. 2005; 18(2):256-9.
   DOI: 10.1111/j.1432-2277.2004.00027
9. Blom KB, Bergo KK, Espe EKS, Rosseland V, Grøtta OJ, Mjøen G, et al. Cardiovascular remodelling in living kidney donors with reduced glomerular filtration rate: rationale and design of the CENS study. Blood Press. 2020;29(2):123-134.
   DOI: 10.1080/08037051.2019.1684817
10. Dounousi E, Mitsis M, Naka KK, Pappas C, Lakkas L, Harisis C, Pappas K, Koutlas V, Tzalavra I, Spanos G, Michalis LK. Differences in cardiac structure assessed by echocardiography between renal transplant recipients and chronic kidney disease patients. In Transplantation ProceedingsElsevier. 2014;46(9):3194-3198.
11. Borrego Utiel FJ, Ramírez Navarro AM, Esteban de la Rosa R, Bravo Soto JA. Comparison of MDRD and the old CKD-EPI equations with the new CKD-EPI equations in kidney transplant patients when glomerular filtration rate is measured with 51Cr-EDTA. Nefrologia (Engl Ed). English, Spanish. 2020;40(1): 53-64.
    DOI: 10.1016/j.nefro.2019.07.006
12. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015; 28(1):1-39.e14.
    DOI: 10.1016/j.echo.2014.10.003
13. Devereux RB, Alonso DR, Lutas EM, Gottlieb GJ, Campo E, Sachs I, Reichek N. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol. 1986; 57:450–458.
14. Otterstad JE, Froeland G, St John SM, Holme I. Accuracy and reproducibility of biplane two- dimensional echocardiographic measurements of left ventricular dimensions and function. Eur Heart J. 1997;18:507–513.

15. Lang RM, Bierig M, Devereux RB, Flaschskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr. 2005;18:1440–1463.
16. Burlacu A, Iftene A, Jugrin D, Popa IV, Lupu PM, Vlad C, Covic A. Using Artificial Intelligence Resources in Dialysis and Kidney Transplant Patients: A Literature Review. Biomed Res Int. 2020;2020: 9867872.
DOI: 10.1155/2020/9867872
17. Vaios V, Georgianos PI, Liakopoulos V, Agarwal R. Assessment and Management of Hypertension among Patients on Peritoneal Dialysis. Clin J Am Soc Nephrol. 2019;14(2):297-305.
DOI: 10.2215/CJN.07480618. Epub 2018 Oct 19
18. Roumeliotis S, Eleftheriadis T, Liakopoulos V. Is oxidative stress an issue in peritoneal dialysis? Semin Dial. 2019; 32(5):463-466.
DOI: 10.1111/sdi.12818. Epub 2019 May 1
19. Jegatheesan D, Cho Y, Johnson DW. Clinical Studies of Interventions to Mitigate Cardiovascular Risk in Peritoneal Dialysis Patients. Semin Nephrol. 2018;38(3):277-290.
DOI: 10.1016/j.semnephrol.2018.02.007
20. Temimović R, Rašić S, Džubur A. Cardiovascular remodelling in patients with pre-dialysis chronic kidney disease and renal transplant recipients. Med Glas (Zenica). 2019;16(2).
DOI: 10.17392/1009-19
21. Alexandrou ME, Sarafidis P, P Theodorakopoulou M, Sachpekidis V, Papadopoulos C, Loutradis C, et al. Cardiac geometry, function, and remodeling patterns in patients under maintenance hemodialysis and peritoneal dialysis treatment. Ther Apher Dial. 2022;26(3):601-612.
DOI: 10.1111/1744-9987.13732.
22. Hawwa N, Shrestha K, Hammadah M, Yeo PSD, Fatica R, Tang WHW. Reverse Remodeling and Prognosis Following Kidney Transplantation in Contemporary Patients With Cardiac Dysfunction. J Am Coll Cardiol. 2015; 66(16):1779-1787.
DOI: 10.1016/j.jacc.2015.08.023
23. Yang M, Miller PJ, Case BC, Gilbert AJ, Widell JK, Rogers T, et al. Pre-Operative Cardiovascular Testing and Post-Renal Transplant Clinical Outcomes. Cardiovasc Revasc Med. 2019;20(7): 588-593.
DOI: 10.1016/j.carrev.2019.04.017. Epub 2019 Apr 17
24. Malyala R, Rapi L, Nash MM, Prasad GVR. Pre-Transplant Left Ventricular Geometry and Major Adverse Cardiovascular Events After Kidney Transplantation. Ann Transplant. 2019; 24:100-107.
DOI: 10.12659/AOT.913649.
25. Hewing B, Dehn AM, Staeck O, Knebel F, Spethmann S, Stangl K, et al. Improved Left Ventricular Structure and Function After Successful Kidney Transplantation. Kidney Blood Press Res. 2016;41(5):701-709.
DOI: 10.1159/000450559
26. Hundae A, McCullough PA. Cardiac and renal fibrosis in chronic cardiorenal syndromes. Nephron Clin Pract. 2014;127(1-4):106-12.
DOI: 10.1159/000363705
27. Wang AY, Lai KN. The importance of residual renal function in dialysis patients. Kidney Int. 2006;69(10):1726-32.
DOI: 10.1038/sj.ki.5000382
28. Chen KT, Kang YN, Lin YC, Tsai IL, Chang WC, Fang TC, et al. Efficacy and Safety of Mineralocorticoid Receptor Antagonists in Kidney Failure Patients Treated with Dialysis: A Systematic Review and Meta-Analysis. Clin J Am Soc Nephrol. 2021;16(6):916-925.
DOI: 10.2215/CJN.15841020
29. Cruzado JM, Pascual J, Sánchez-Fructuoso A, Serón D, Díaz JM, Rengel M, et al.; Evita Study Group. Controlled randomized study comparing the cardiovascular profile of everolimus with tacrolimus in renal transplantation. Transpl Int. 2016;29(12):1317-1328.
DOI: 10.1111/tri.12862
30. Garcia-Covarrubias L, Hernández K, Castro I, Hinojosa H, Molina L, Bazan O, et al. Cardiac Remodeling in Structure and Function Six Months After Kidney Transplantation. Transplant Proc. 2018; 50(2):454-457.
DOI: 10.1016/j.transproceed.2017.12.038.

31. Joseph MS, Tinney F, Naik A, Parasuraman R, Samaniego-Picota M, Bhave NM. Right Ventricular Dysfunction and Adverse Outcomes after Renal Transplantation. Cardiorenal Med. 2021;11(2):109-118.
DOI: 10.1159/000515124.



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