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Improvement in left ventricle geometry and function after kidney transplantation

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ABSTRACT

Chronic kidney disease (CKD) is associated with remodeling of the left ventricle (LV), affecting both its geometry and function. Kidney transplantation in patients with stage 5 CKD may lead to improvements in LV remodeling and result in better cardiac function. The aim of the study was to determine changes and improvements in LV geometry and function after kidney transplantation in patients with stage 5 CKD. This was an observational study conducted by collecting secondary data from the Hospital's Kidney Transplantation Registry, Dr. Sardjito General Hospital spanning the years 2017 to 2020. The study employed a comparative design, contrasting the results before and after treatment (kidney transplantation). We compared transthoracic echocardiographic parameters for LV geometry and function before and after kidney transplantation. The evaluation timeframe after kidney transplantation was divided into <12 and ≥12 mo. A total of 27 patients qualified for inclusion in this study. In the <12 mon (n=20) evaluation group, there was a reduction in proportion of LV hypertrophy from 70% to 45%. There was an increase in global LV systolic function (ejection fraction) from 60.1±10.95% to 67.85±6.48% (p=0.014), and a decrease in LV diastolic dysfunction from 45% to 15% (p=0.07). In the ≥12 mon (n=11) evaluation group, there was a decrease in the proportion of LV hypertrophy from 81.8 to 54.5%, an increase in global LV systolic function (ejection fraction) from 57.73±13.07% to 69.36±6.12% (p=0.011), and a decreased LV diastolic dysfunction from 63.6% to 0% (p=0.016). In conclusion, significant changes in LV geometry and function are observed following kidney transplantation, indicating improvements in these parameters. There are improvements in LV systolic function started at <12 mo and in LV diastolic function at ≥12 mo after kidney transplantation.

ABSTRAK

Penyakit ginjal kronis (PGK) dikaitkan dengan remodeling ventrikel kiri (VK), yang mempengaruhi geometri dan fungsinya. Transplantasi ginjal pada pasien dengan PGK stadium 5 dapat menyebabkan perbaikan dalam remodeling ventrikel kiri (VK) dan menghasilkan fungsi jantung yang lebih baik. Tujuan penelitian adalah untuk mengetahui perubahan dan perbaikan geometri dan fungsi VK setelah transplantasi ginjal pada pasien PGK stadium 5. Ini adalah penelitian observasional yang dilakukan dengan mengumpulkan data sekunder dari Registrasi Transplantasi Ginjal Rumah Sakit, RSUP Dr. Sardjito, Yogyakarta selama tahun 2017 hingga 2020. Penelitian ini menggunakan desain komparatif, yang membandingkan hasil sebelum dan sesudah perawatan (transplantasi ginjal). Kami membandingkan parameter ekokardiografi transthorak untuk geometri dan fungsi VK sebelum dan sesudah transplantasi ginjal. Jangka waktu evaluasi setelah transplantasi ginjal dibagi menjadi <12 dan ≥12 bulan. Sebanyak 27 pasien memenuhi syarat untuk diikutsertakan dalam penelitian ini. Pada kelompok evaluasi <12 bulan (n=20), terdapat penurunan proporsi hipertrofi ventrikel kiri dari 70% menjadi 45%. Terdapat peningkatan fungsi sistolik VK global (fraksi ejeksi) dari 60,1±10,95% menjadi 67,85±6,48% (p=0,014), dan penurunan disfungsi diastolik VK dari 45% menjadi 15% (p=0,07). Pada kelompok evaluasi ≥12 bulan (n=11), terjadi penurunan proporsi hipertrofi ventrikel kiri dari 81,8 menjadi 54,5%, peningkatan fungsi sistolik ventrikel kiri global (fraksi ejeksi) dari 57,73±13,07% menjadi 69,36±6,12% (p=0,011), dan penurunan disfungsi diastolik VK dari 63,6% menjadi 0% (p=0,016). Kesimpulannya, perubahan signifikan pada geometri dan fungsi VK diamati setelah transplantasi ginjal, yang menunjukkan perbaikan pada parameter ini. Terdapat perbaikan pada fungsi sistolik VK yang dimulai pada <12 bulan dan fungsi diastolik LV pada ≥12 bulan setelah transplantasi ginjal.

Keywords:
kidney transplantation; left ventricular function; left ventricular geometry; chronic kidney disease; cardiopathy

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INTRODUCTION

Cardiovascular complications can manifest at any stage of chronic kidney disease (CKD), irrespective of the glomerular filtration rate (GFR).[1] Left ventricular hypertrophy (LVH) stands as a hallmark of uremic cardiopathy, closely associated with type 4 cardiorenal syndrome or chronic cardiorenal syndrome, a consequence of CKD. Left ventricular hypertrophy arises from chronic pressure or volume overload, resulting in increased cardiac wall pressure.[1] In its early stages, it is deemed an adaptive response to these overloads. Notably, left ventricular diastolic filling disturbances are frequently observed.[2] Subsequently, the remodeling of LV geometry persists, eventually leading to the disruption of left ventricular systolic function.[2]

Stage 5 CKD, also known as end-stage renal disease (ESRD) necessitates kidney replacement therapy. Three modalities are available: hemodialysis, peritoneal dialysis, and kidney transplantation. According to the 2018 Indonesian Renal Registry (IRR) data in the 11th IRR Report, the majority of services provided at dialysis service facilities are hemodialysis (98%), while continuous ambulatory peritoneal dialysis (CAPD) services make up 2%.[3] Furthermore, as much as 11% of CAPD patients have discontinued and transferred to hemodialysis, with kidney transplantation accounting for as much as 1%.[3]

Kidney transplantation is associated with improvements in cardiac structure and function.[4] For patients with stage 5 CKD, kidney transplantation yields positive cardiovascular effects and enhances cardiac function.[5] Left ventricular global function was assessed by measuring the difference between end-diastolic and end-systolic values of a one-dimensional (1D), 2D, or 3D parameter divided by the end-diastolic value.[6] In a previous study, we found that among 15 kidney transplant recipients, left ventricular ejection fraction (LVEF) significantly increased, with a mean improvement of 14.3% after kidney transplantation. Notably, all patients with reduced ejection fraction exhibited an increase in LVEF, with a mean improvement of 37%. The proportion of patients with diastolic dysfunction decreased significantly.[7] In this study, we expand and confirm these findings by including more subjects. Our aim is to investigate the impact of kidney transplantation on left ventricle geometry and function, which will be assessed before and after kidney transplantation using transthoracic echocardiography (TTE).

MATERIAL AND METHODS

Design and subjects

This is an observational study conducted by collecting secondary data from the Hospital Kidney Transplantation Registry of Dr. Sardjito General Hospital, Yogyakarta. The study used a comparative design comparing the before and after treatment (kidney transplantation) result. This study was performed after obtaining ethical clearance from the Medical and Health Research Ethics Committee of the Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada Yogyakarta, Indonesia (No: KE/FK/0804/EC/2021).

This study enrolled subjects who were kidney transplant recipients at Dr. Sardjito General Hospital, from August 2017 until December 2020. The inclusion criteria were patients with 1) aged >18 yr; 2) underwent kidney transplantation and recorded in the kidney transplant registry of our hospital; 3) underwent TTE examination within 3 mo before transplantation; and 4) underwent TTE examination after transplantation. The exclusion criteria were the incomplete data required for primary outcome analysis, i.e. TTE data.

Procedures

Trained sonographers from the echocardiography division conducted the TTE using one of the echocardiography machines: Vivid 7 (GE Vingmed, Norway, M4s transducer), Vivid 6 (GE Vingmed, Norway, M4s transducer), T8 (GE Vingmed, China, 3Sc transducer), Epiq 7 (Phillips, USA, X5-1 transducer), or E95 (GE Vingmed, Norway, M5Sc transducer). The baseline TTE was performed within 3 mo before kidney transplantation, while the evaluation TTE was conducted after the transplantation. Intraobserver and interobserver validity tests had been previously carried out.[8] The TTE parameters recorded during the examination were based on standard parameters recommended by the Indonesian Society of Echocardiography, following international guideline.[2]

Measurements

The left ventricular geometry was measured based on the M-mode linear method. It was obtained from the parasternal long axis approach and perpendicular to the long axis of the left ventricle, and were measured at the height of the leaflet tip of the mitral valve during the end diastolic phase. The left ventricle mass index (LVMi) and relative wall thickness (RWT) values were obtained, and categorized into 1) normal (male: LVMi ≤115 g/m^2, RWT ≤0.42; female: ≤95 g/m^2, RWT ≤0.42); 2) left ventricle concentric remodeling (male: LVMi ≤115 g/m^2, RWT >0.42; female: ≤95 g/m^2, RWT >0.42); 3) left ventricle concentric hypertrophy (male: LVMi >115 g/m^2, RWT >0.42; female: >95 g/m^2, RWT >0.42); or 4) left ventricle eccentric hypertrophy (male: LVMi >115 g/m^2, RWT ≤0.42; female: >95 g/m^2, RWT ≤0.42).[2] The left atrial volume index (LAVI) was measured based on 2D biplane method.[2]

The left ventricular functions investigated were systolic and diastolic functions. Assessment of systolic function was based on 2D biplane or linear M-mode method. The 2D biplane method was obtained from the modified Simpson method approach. The left ventricular systolic dysfunction was determined as LVEF ≤50%. Assessment of diastolic function was done based on 4-variable methods using pulse wave Doppler for assessment of E, A, e' wave velocity and continuous wave Doppler for tricuspid regurgitant jet velocity assessment.[2]

Statistical analysis

Statistical analysis using SPSS software v.26 (IBM Corp., USA). The comparison and changes of TTE parameters, as numerical data, before and after kidney transplantation were tested by paired t-tests and Wilcoxon tests, where applicable. The Kolmogorov-Smirnov or Shapiro-Wilk tests was conducted to determine the distribution of numerical data. The categorical data were compared by McNemar test. The subjects were divided into groups based on the period of TTE evaluation performed at follow-up, namely the evaluation group <12 mo (n=20 subjects) and the evaluation group ≥12 mo (n=11 subjects). The p value <0.05 was set as a guide for statistical significance.

RESULTS

Subjects Characteristics

A total of 27 subjects qualified for this study. The mean age among these subjects was 37.67±12.35 y.o., with male subjects comprising the highest proportion (n=18, 66.7%). The mean duration of hemodialysis was 16.0 (range: 4.0-84.0) months. Comorbidities included hypertension (n=17, 63.0%) and diabetes mellitus (n=5, 18.5%). TABLE 1 shows the subjects characteristics.

The changes in left ventricular

geometry, as well as systolic and diastolic functions before and after kidney transplantation, are presented in TABLE 2. Significant improvements were observed in left ventricle geometry, with a reduction in the proportion of LV eccentric hypertrophy, transitioning towards milder LV remodeling. Both LV systolic and diastolic functions exhibited improvement following kidney transplantation. Notably, LVEF increased post-translation. Additionally, LAVI demonstrated improvement after kidney transplantation. Laboratory parameters also exhibited positive changes, with hemoglobin levels and creatinine levels notably improving.

TABLE 3 presents the analysis of the changes in LV geometry, systolic and diastolic functions before and during two different post-kidney transplantation follow-up periods, namely <12 mo and ≥12 mo. Twenty subjects were observed during the <12 mo follow-up period after kidney transplantation. LVEF significantly increased after kidney transplantation (p=0.014). Four subjects with LV systolic dysfunction before kidney transplantation exhibited improved function after transplantation. Eight subjects with diastolic dysfunction prior to kidney transplantation showed better function after transplantation, with only one subject still presenting diastolic dysfunction. Within the <12 mo follow-up period after kidney transplantation, LV geometry improved, as indicated by a reduction in LV eccentric hypertrophy and an increase in LV concentric remodeling.

Eleven subjects were observed until ≥12 mo follow-up. The LVEF was significantly increased (p=0.011). Three subjects with LV systolic dysfunction before kidney transplantation had improved LV systolic function after transplantation. Seven subjects with diastolic dysfunction before kidney transplantation, all had improved after transplantation. The LV geometry shifted into improvement in ≥12 mo follow-up after kidney transplantation, indicated by reduced LV eccentric hypertrophy and increased LV concentric remodeling.

Before kidney transplantation, there were two subjects with LVEF <40%, due to global hypokinetic, and within <12 mo follow-up after kidney transplantation, the LVEF improved into normal value. Two other subjects with LVEF <40%, one with global hypokinetic and another with segmental hypokinetic, were evaluated ≥12 mo follow-up and became normal ejection fraction (FIGURE 1). These four subjects with LVEF <40% were males, aged between 26 to 52 yr, had hemodialysis duration from 13 to 84 mo. All had hypertension and none had diabetes mellitus.

TABLE 1. The comparison of the characteristics of subjects during pre-and post-kidney transplantation periods

Characteristics	Pre-transplantation (n=27)	Post-transplantation (n=27)	p
Age (mean ± SD yr)	37.67±12.35	37.67±12.35	NA
Males [n (%)]	18 (66.7)	18 (66.7)	NA
Hemodialysis duration (mo)*	16.0 (4.0-84.0)	16.0 (4.0-84.0)	NA
Hypertension [n (%)]	17.0 (63.0)	17.0 (63.0)	NA
Use of antihypertension [n (%)]	17 (100)	12 (70.6)	0.063
Diabetes mellitus [n (%)]	5 (18.5)	5 (18.5)	NA

*Data were expressed as median (range value).

TABLE 2. The comparison of the changes in left ventricular geometry and functions during pre- and post-kidney transplantation periods

Characteristics	Pre-transplantation (n=27)	Post-transplantation (n=27)	p
LV Geometry [n (%)]			0.038
• Normal geometry	6 (22.2)	5 (18.5)	
• LV eccentric hypertrophy	10 (37.0)	0 (0)	
• LV concentric hypertrophy	10 (37.0)	12 (44.4)	
• LV concentric remodeling	1 (3.7)	10 (37.0)	
LV ejection fraction (mean ± SD %)	59.22±12.3	69.26±5.95	<0.001
LV systolic dysfunction [n (%)]	7 (25.9)	0 (0.0)	0.016
LV diastolic dysfunction [n (%)]*	12 (44.4)	0 (0.0)	0.002
LAVI [med (min–max) mL/m^2]	39.0 (16.0-82.0)	24.0 (14.0-51.0)	<0.001
LVIDd (mean ± SD mm)	51.85±6.7	43.52±5.89	<0.001
Hemoglobin (mean ± SD g/dL)	9.78±1.85	14.06±2.08	<0.001
Urea nitrogen [med (min-max)mg/dL]*	11.8 (4.2-44.6)	18.2 (5.4-50.9)	0.011
Creatinine [med (min-max) mg/dL]*	2.91 (1.30-6.13)	1.28 (0.76-3.09)	<0.001

*1 subject can not be evaluated; LV: left ventricle; LAVI: left atrial volume index; LVIDd: left ventricle internal diameter end diastole; NA: not applicable; SD: standard deviation; med: median; min: minimum; max: maximum

TABLE 3. The changes in LV geometry and functions between pre- and post-kidney transplantation in the follow-up evaluation period of <12 and ≥ 12 mo

Evaluation periods/Parameters	Pre-transplantation	Post-transplantation	p
Evaluation periods of < 12 mo (n=20)			
LV ejection fraction (mean ± SD %)	60.1±10.95	67.85±6.48	0.014
LV systolic dysfunction [n (%)]	4 (20)	0 (0)	0.125
LV geometry [n (%)]			
• Normal	5 (25.0)	3 (15.0)	0.135
• LV eccentric hypertrophy	6 (30.0)	0 (0.0)	
• LV concentric hypertrophy	8 (40.0)	9 (45.0)	
• LV concentric remodeling	1 (5.0)	8 (40.0)	
LV diastolic function [n (%)]*			
• Normal	11 (55.0)	17 (85.0)	0.061
• Indeterminate	0 (0.0)	1 (5.0)	
• Diastolic dysfunction	8 (40.0)	1 (5.0)	
Evaluation periods of ≥12 mo (n=11)			
LV ejection fraction [mean ± SD %]	57.73±13.07	69.36±6.12	0.011
LV systolic dysfunction [n (%)]	3 (27.3)	0 (0)	0.250
LV geometry [n (%)]*			
• Normal	2 (18.2)	3 (27.3)	0.116
• LV eccentric hypertrophy	5 (45.5)	0 (0.0)	
• LV concentric hypertrophy	4 (36.4)	6 (54.4)	
• LV concentric remodeling	0 (0.0)	2 (18.2)	
LV diastolic function [n (%)]			
• Normal	4 (36.4)	11 (100.0)	0.016
• Diastolic dysfunction	7 (63.6)	0 (0.0)	

*1 subject could not be evaluated, SD: standard deviation.

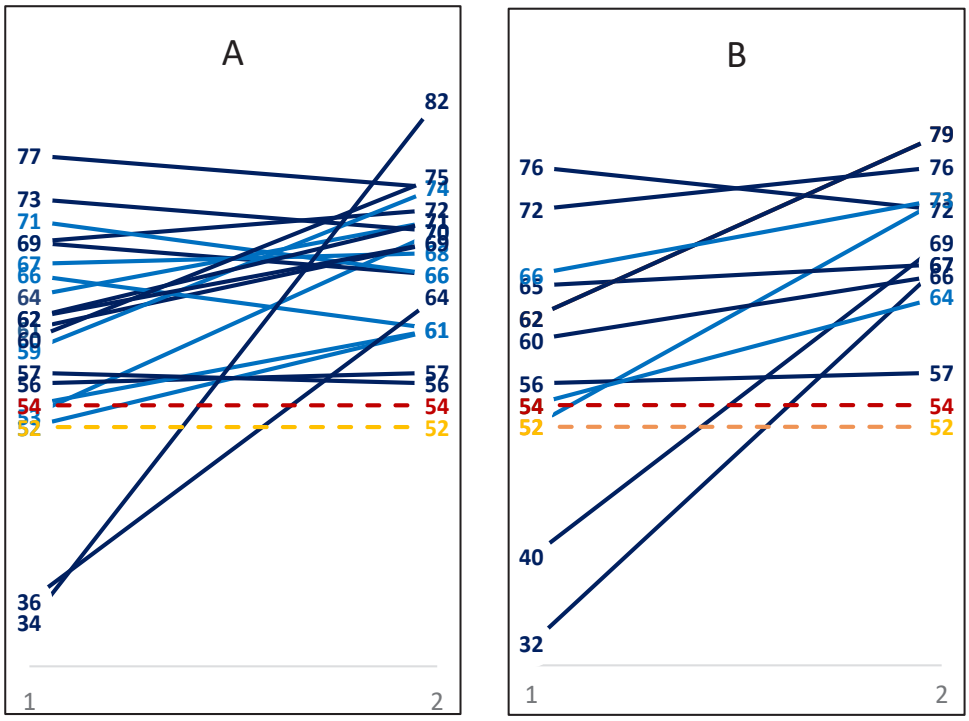

FIGURE 1. The graph of the changes in LV ejection fraction. A) The evaluation within <12 mo follow-up post-kidney transplantation (n=20). B) The evaluation ≥12 mo follow-up post-kidney transplantation (n=11). 1) Pre-kidney transplantation; 2. Post-kidney transplantation. Dark blue line: males. Light blue Line: females. Red dotted line: female LV ejection fraction normal limit. Yellow dotted line: male LV ejection fraction normal limit.

TABLE 4 shows the comparison of TTE parameters, diagnosis and laboratory results between before and after kidney transplantation within evaluation of <12 mo follow-up and ≥12 mo follow-up periods. In subjects with evaluation <12 mo follow-up, there were significant changes in most of TTE parameters namely RWT, LVMi, LAVI, LV diameters, LV ejection fraction, E/A ratio and tricuspid regurgitation velocity. These significant changes were also observed in subjects with ≥12 mo follow-up, except for RWT and E/A ratio. The proportion of subjects with LV hypertrophy tended to decrease after kidney transplantation in both evaluation groups. Subjects with systolic and diastolic dysfunctions were improved after kidney transplantation in both evaluation groups. There were also significant changes in laboratory values after kidney transplantation.

TABLE 4. The TTE parameters, diagnosis and laboratory results between pre- and post-kidney transplantation in evaluation period of <12 and ≥12 mo follow-up

Parameters	<12 mo follow-up (n=20)		p	≥12 mo follow-up (n=11)		p
	Pre-transplantation	Post-transplantation		Pre-transplantation	Post-transplantation	
TTE parameters						
• Relative wall thickness [med (min – max)]	0.42 (0.32-0.97)	0.5 (0.39-1.03)	0.030	0.38 (0.34-0.97)	0.47 (0.33-0.67)	0.328
• LV mass index (mean ± SD g/m^2)	154.0±61.0	116.0±36.9	0.004	172.4±61.9	96.6±28.7	0.006
• LAVI (mean ± SD mL/m^2)	38.0 (16.0-82.0)	22.5 (12.0-51.0)	0.001	51.9±17.7	25.0±7.1	<0.001
• LVIDd (mean ± SD mm)	50.95±7.2	43.4±5.56	<0.001	53.91±7.44	43.73±6.21	<0.001
• LV ejection fraction (mean ± SD %)	60.1±10.95	67.85±6.48	0.014	57.73±13.1	69.36±6.12	0.011
• E/A ratio [median (min – max)]	1.2 (0.78-3.10)	1.03 (0.6-3.0)	0.018	1.3 (0.9-92.0)	0.79 (0.0-9.0)	0.075
• TR velocity [median (min – max) m/s]	1.2 (0.0-4.79)	0.0 (0.0-2.76)	0.028	2.74 (0.0-3.96)	0.0 (0.0-2.45)	0.018
Diagnosis						
• LV hypertrophy [n (%)]	14 (70.0)	9 (45.0)	0.125	9 (81.8)	6 (54.5)	0.375
• Systolic dysfunction [n (%)]	4 (20.0)	0 (0.0)	0.125	3 (27.3)	0 (0.0)	0.250
• Diastolic dysfunction [n (%)]	9 (45.0)	3 (15.0)	0.070	7 (63.6)	0 (0.0)	0.016
Laboratory results						
• Hemoglobin (mean ± SD g/dL)	9.8±1.68	13.33±2.27	<0.001	9.6±2.1	14.66±2.01	<0.001
• Urea nitrogen (mean ± SD mg/dL)	14.35 (4.2-44.6)	18.8 (10.4-50.9)	0.121	8.5 (4.2-23.5)	18.4 (5.4-30.9)	0.021
• Creatinine (mean ± SD mg/dL)	3.11 (1.30-6.13)	1.13 (0.76-3.09)	<0.001	2.57±0.85	1.45±0.42	0.001

SD: standard deviation; med: median; min: minimum; max: maximum; TEE: transthoracal echocardiography; LV: Left ventricle; LAVI: Left atrial volume index, LVIDd: Left ventricle internal diameter end diastole; TR: tricuspid regurgitation.

DISCUSSION

The results of our study indicated that there were improvements in LV geometry, LV systolic and LV diastolic function after kidney transplantation. The improvement in LV geometry tended to occur within one year after kidney transplantation and continued after one-year follow-up. The proportion of LV hypertrophy tended to reduce. The LV systolic dysfunction improved in one-year evaluation and more than one-year follow-up. The increase in LVEF was significantly raised to within the normal range subjects with initially reduced LVEF. Improved LV diastolic function was observed in the one-year evaluation and continued to be significant one year after kidney transplantation.

Previous studies have indicated changes in LV structure and geometry based on echocardiographic examinations in patients with stage-V-CKD at the initiation of hemodialysis therapy.[9] In patients who underwent kidney transplantation, the changes in LV structure and geometry were observed in previous studies. The comparison of echocardiographic results in the

before kidney transplantation and after kidney transplantation periods showed significant improvement changes.[10] Our study indicated similar findings such that the LV geometry and functions corrected significantly after kidney transplantation among stage V CKD patients.

Our study demonstrated significant improvements in LV systolic function within one year, which were sustained in the one-year evaluation. These favorable changes in systolic function after kidney transplantation at 6 and 12 mo were observed by other previous study.[5] The kidney transplantation can be performed safely in stage 5 CKD patients with decreased LV ejection fraction, advanced heart failure, and without induced ischemia.[11] Our study indicated that subjects with LV ejection fraction <40% had returned into normal ejection fraction after kidney transplantation. Improved global systolic function after kidney transplantation is associated with a reduction in excessive blood flow (preload), resulting in enhanced left ventricular inotropic function.[12] The duration of hemodialysis before kidney transplantation is a significant factor that predicts the normalization of LVEF.[11]

The analysis of LV diastolic function showed a significant improvement after one year of kidney transplantation, with the trend toward improvement within one-year follow-up. In the previous study, the diastolic dysfunction did not change significantly after 12 mo of kidney transplantation.[13] This is associated with pathological changes in myocardial anatomy, such as myocardial calcification or fibrosis, which may lead to a lack of improvement in the diastolic index after kidney transplantation.[13] The worsening of LV diastolic function may be attributed to the use of cyclosporine post-transplantation and inadequate blood pressure control.[12]

There was a trend toward the resolution of LV hypertrophy after kidney transplantation, both within the first year and in the follow-up after one yr. The non-significant decrease in LV hypertrophy may be related to the persistence of hypertension that occurs among subjects. We observed the persistence of hypertension after kidney transplantation, with as many as 70.6% of hypertensive subjects continuing to consume anti-hypertensive therapy. It is possible that the persistence of hypertension was due to hypertension being the primary comorbid condition in the subjects, or another possibility of LV hypertrophy associated with treatment using calcineurin inhibitors and steroids. Calcineurin inhibitors and steroids may induce hypertension. All subjects in our study used calcineurin inhibitor (tacrolimus) and steroids as immunosuppressant therapies.

The results of our study showed different findings from other previous studies, which found significant improvement in LV geometry after kidney transplantation. Successful kidney transplantation improves some risk factors for LV hypertrophy such as uremia, anemia, and hyperparathyroidism, but other factors such as patent arteriovenous fistula, dyslipidemia, and hypertension may persist or even worsen after kidney transplantation. Immunosuppressive drugs, such as calcineurin inhibitors, especially cyclosporine and steroids that have the side effect of hypertension play an important role in the development or persistence of LV hypertrophy after kidney transplantation.[10] The presence of an arteriovenous fistula reduces peripheral resistance, thereby creating a hyperkinetic circulation, which increases heart rate and stroke volume index.[12] Changes in LV geometry have been associated with improved blood pressure control, reflecting the impact of afterload on left ventricular remodeling.[2] Our study revealed a wide range of ages among participants; however, age did not appear to affect the outcome of

kidney transplantation.

Uremic toxins in plasma have negative inotropic and chronotropic potential[14] and prolonged exposure to these uremic toxins can cause myocyte fibrosis and death.[15,16] These reasons may suggest that the duration of dialysis can affect changes in diastolic function. Prolonged exposure to uremic toxins has been shown to affect myocardial contractility so that the possibility of improvement in systolic function can also be affected by the duration of dialysis.[17,18] In our study, the mean duration of hemodialysis before kidney transplantation procedure was 16.0 mo.

The kidney transplant graft dysfunction and sustained high blood pressure may contribute to the lack of improvement in LV hypertrophy and other cardiovascular risk factors, such as increased extracellular volume, electrolyte abnormalities, malnutrition, anemia, and uremic toxins, after kidney transplantation.[13] Systolic blood pressure was associated with LV mass and LV mass index at 12 mo after kidney transplantation.[13] There was a significant reduction in LV hypertrophy after kidney transplantation. One explanation for this effect is the possible decrease in systolic blood pressure and improvement in kidney function after kidney transplantation. This notion demonstrates the importance of graft function in the development of LV hypertrophy after kidney transplantation.[13] Insulin resistance, metabolic syndrome, and type 2 diabetes mellitus are associated with increased LV mass, RWT, and diastolic dysfunction.[18] Patients with diabetes mellitus also tend to have decreased systolic function.[12,18] Therefore, the role of comorbidities is also an important factor affecting the LV geometry and function among kidney transplant patients.

There were several limitations identified in this study. First, the small number of subjects from a single center needed to be corroborated by a larger number of study subjects from a multicenter national registry. Two, the relatively short period of observation and follow-up due to the nature of study design needed to be corroborated with a longer and continuous evaluation by using a prospective cohort design. Three, the adequacy of hemodialysis before and technical difficulties during kidney transplantation procedure were not reported in detail in this study.

CONCLUSION

The LV geometry, LV systolic and LV diastolic functions improve after kidney transplantation. The improved LV geometry tends to occur within one year after kidney transplantation and continues after one year follow-up. The LV systolic dysfunction improved in one year evaluation and more than one year follow-up. The LVEF fraction increases significantly even in those with LVEF <40%. The LV diastolic function significantly recovers after one year after kidney transplantation.

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