Structural and functional echocardiographic abnormalities in children pre- and post-kidney transplant in Cape Town, South Africa

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Summary

Introduction: Cardiovascular manifestations of chronic kidney disease (CKD) include structural changes such as left ventricular hypertrophy (LVH) related to hypertension and fluid overload, vascular wall thickening, and calcification. There are also functional abnormalities such as systolic and diastolic dysfunction including - reduced ejection fraction (EF) and fractional shortening (FS), diastolic dysfunction, arrhythmias, and increased stiffness of vessels. The risk of these abnormalities worsens with a decreasing glomerular filtration rate (GFR).

The best treatment for patients with kidney failure is kidney transplantation. Candidates are usually screened for cardiac abnormalities, and it is not uncommon to find echocardiographic abnormalities.

It is important to study how patients with cardiac abnormalities on echocardiography (echo) do during and after kidney transplantation to optimize pre-transplant treatment, peri-transplant care, and short and long-term post-transplant care.

Objectives: The objective of this study is to identify patients with KF who had structural and functional cardiac abnormalities on echo before kidney transplant and to determine whether these abnormalities improved or worsened post-transplant.

Method: Single-center retrospective review of children with cardiac abnormalities on echo who had kidney transplants from 1st January 2018 to 31st Dec 2023 at Red Cross War Memorial Children's Hospital (RCWMCH).

Result: Thirty-two patients out of forty-three (74%) had echocardiographic screening within one year before kidney transplant. Out of these 15/32 (46.9%) had abnormal structural or functional echocardiographic findings before transplant. Of the abnormal echocardiography, left ventricular systolic and diastolic constitute 6/32 (19%) while 3/15(9%) had left ventricular hypertrophy. During the study period, a total of 5 patients died after transplant (11.6%). There was no significant difference in independent variables between survivors and those who died (P value>0.05). There was no significant difference between ejection fraction ($p = 0.5$) or shortening fraction ($p = 0.4$) pre- to post-transplant, in those who had paired samples (n=6) [using Wilcoxon signed rank test]. Similarly, there was no association between pre-transplant ejection fraction or fractional shortening and immediate graft functioning or graft rejection ($p > 0.1$).

Conclusion: Our study showed that close to half of patients who had echocardiographic screening had abnormality. Left ventricular systolic and diastolic dysfunction were the more common compared to left ventricular hypertrophy.There was no significant difference between ejection fraction and fractional shortening on echocardiographic evaluation pre and post-transplant among 6 patients who had paired pre and post-transplant echocardiographic study.

Keywords: Structural and functional cardiac abnormalities, kidney transplant

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1. Introductions

1.1. Background

The kidneys are responsible for many physiological processes, including clearance of metabolic waste products of the body, salt and water homeostasis, regulation of blood pressure, and hormone production. Cardiovascular disease in chronic kidney disease (CKD) can be due to hypertension and fluid overload mediated by salt and water retention, sympathetic overactivity, activation of the renin-angiotensin system (RAAS), and accumulation of endogenous vasopressors. In turn, hypertension can damage the kidneys further, leading to a vicious cycle of rising blood pressure with arterial hyalinosis, vascular stiffening, and declining glomerular filtration rate (GFR) (1).

The mechanism by which cardiovascular disease develops in children with CKD is thought to occur by cardiac remodeling and vascular injury that occur in CKD. Remodeling occurs due to left ventricular hypertrophy (LVH) from increased afterload caused by vascular stiffness or haemodynamic overload from high output states like anaemia.As CKD progress worsening LVH may lead to decreased sub-endothelial myocardial perfusion and an increased risk of cardiac rhythm disturbances. Persistent hypo-perfusion may finally lead to myocardial fibrosis and diastolic or systolic dysfunction(1).

Changes in the cardiac geometry are initially adaptive changes to volume and pressure stress overload. Volume overload increases left ventricular filling pressure and thereby stretches the ventricular wall. The heart adapts by increasing the length existing myocytes, thus enlarging the dimension of the left ventricle. The increased left ventricular volume is usually accompanied by wall thickening decreasing wall stress further. Thus adaptation to volume overload results in a ventricle with a thick wall with enlarged cavity, but the left ventricular wall thickness-to-internal diameter ratio remains normal (*eccentric hypertrophy*).Clinically eccentric left ventricular hypertrophy usually accompanies volume overload, anemia, and arteriovenous shunt (2).

In contrast, pressure overload increases wall stress during systole leads to myocyte hypertrophy and wall thickening, left ventricular cavity volume remains preserved (*concentric hypertrophy*). There is a parallel addition of sarcomeres increasing the cross-sectional area and diameter of the myocytes. Concentric hypertrophy is associated with systolic or pulse pressure.

These adaptive changes may be reversible in the early stages and are initially beneficial. Dilation permits increased cardiac output(CO) for a similar level of energy expenditure, whereas wall thickening redistributes increased wall tension over a larger area and decreases energy consumption per myocytes(2).

At the start LVH is adaptive and beneficial. It optimizes ejection fraction by normalizing systolic wall stress and reducing tension among a greater number of sarcomeres. Prolonged and proportionally increased pressure and volume overload with declining renal function is accompanied by maladaptive cardiac hypertrophy leading to congestive heart failure. The increased cardiac myocyte work relative to the oxygen supply leads to myocyte death and fibrosis. This eventually leads to systolic dysfunction, diastolic dysfunction and arrhythmias (3)(4).

Mitsnefes et al.on a retrospective study of 64 patients with kidney failure and age range 20 months to 22 years on maintenance dialysis showed the prevalence of LVH was 75%..LVH affected 85% patients on hemodialysis (HD) and 68% on peritoneal dialysis (PD). The prevalence of LVH in patients with HD was significantly higher than those on those on chronic peritoneal dialysis compared to hemodialysis (P=0.02)(5).

Left ventricular dysfunction in children with CKD is different compared to adults with CKD,earlier appearing diastolic dysfunction may be found while earlier systolic dysfunction is typical in adults(6)(7)(8).

Cardiovascular diseases are the leading cause of death in children with kidney failure (9)(10). United States Renal Data System (USRDS) of the study period 2011 to 2020 showed cardiovascular causes are the leading causes of death overall in children with kidney failure(25%), children on HD (31.1%), and PD (31.1%) whereas cardiovascular causes constitute 9.6% of death in children who underwent renal transplant. Cardiac arrest, arrhythmia, and heart failure are leading causes of cardiovascular death in children in contrast to myocardial infarction which most common cause of cardiovascular death in adults with CKD (11).

Numerous studies have shown an inverse association between estimated GFR (eGFR) and cardiovascular risk. In the '4C' (Cardiovascular Comorbidities in Children with CKD study), the prevalence of LVH increased from 10% to 48% as the GFR declined from stage 3 to stage 5 CKD in patients(12).

The Kidney Disease Improving Global Outcomes (KDIGO) guidelines recommend that in children with CKD, anti-hypertensive treatment should be started when the BP is consistently above the 90^{th} percentile for age, sex, and height and to aim for Blood pressure below or equal to the 50^{th} percentile highlighting the important role of hypertension in heart disease in CKD patients especially in the presence of proteinuria(13).

Treatment of anemia of CKD and serum calcium and phosphate are also vital due to growing evidence of their contribution to vascular abnormalities(14).

Ashwin et al showed the improvement in the cardiac function of 6 paediatric patients with cardiac dysfunction who had kidney transplantation between January 2001 and December 2010. The patients age range was 5 to 17 years. Maximum medical support with dialysis and heart failure treatment brought improvement in cardiac function but did not normalize cardiac functions. After kidney transplantation one-third of patients had normalization of cardiac function at discharge while two third had normalization of systolic function during the first year post-transplant. The mean ejection fraction was 22 units better than the pre-transplant value. All patients had excellent graft function in the post-kidney transplant period(15).

Another retrospective study was done on 26 children who underwent kidney transplantation from April 2014 to December 2016 in Japan also showed improvement in LVH post-transplant. Patients were grouped into two based on the presence of left ventricular hypertrophy before kidney

transplant and changes in cardiac function were evaluated 1-year post-transplant. The echocardiographic parameters for LVH and EF were significantly improved 1 year after renal transplant in the group who had LVH initially , and no change was observed in the group not having LVH initially (16).

Cardiovascular death in children and young adults who had kidney failure during childhood are 1,000 times greater than the rates in same aged healthy individuals(17). Cardiovascular mortality in paediatrics with CKD is different from adults. In adult cardiovascular mortalities are frequently from coronary artery disease(CAD) and congestive heart failure(CHF), while the leading causes of cardiac death in children with CKD are arrhythmias and cardiomyopathies(18).

Kidney transplantation is currently the preferred therapy for advanced CKD and kidney failure since it provides better quality of life and extends life expectancy. The CKD cardiovascular relationship continues post- kidney transplant as well, so clinicians have to decrease the risk of significant cardiovascular complications and death by restoring an optimal amount of kidney function. (1)

Since cardiovascular disease remains a major contributor to the overall post-transplant co-morbidity and mortality burden, pre-transplant echocardiographic screening is essential to improving post-transplant outcome. Pre-transplant cardiac screening includes a thorough focused clinical history and examination, electrocardiography(ECG), and echocardiography with further imaging such as cardiac MRI if deemed necessary(19).

In our center patients undergoing kidney transplantation are screened with pre-transplant echocardiography. If the pre-transplant echo is abnormal follow-up echocardiography will be done post-transplant. There is no cut-off EF to turndown patients for kidney transplantation as the hope is that most of the cardiac abnormalities are due to causes like fluid overload and hypertension from CKD and that these will resolve post-transplantation.

In this longitudinal study, we will look at the structural and functional changes in children who had abnormalities before transplantation based on echocardiography.

1.2 Statement of the problem

Children who had normal structure and function before dialysis initiation frequently develop structural and functional abnormalities afterward. The risk of cardiac disease starts with non-dialysis requiring CKD and worsens with the stage of CKD(12). There is a scarcity of data on cardiac changes post-transplantation. In this retrospective study, our purpose is to assess the post-transplant changes in patients diagnosed with cardiac abnormalities before kidney transplantation.

1.3 Significance of the Study

The availability of dialysis and kidney transplantation has brought improvement in the overall outcome of patients with KF. To date, no study has been done in South Africa or Africa to evaluate the function and structure of the heart post-kidney transplantation in paediatric patients who had cardiac abnormalities on echocardiography before transplantation. The results of this study may

help in the pre-transplant optimization of patients with kidney failure, better management of such patients post-transplant, and prioritizing patients for kidney transplantation. Adult patients with kidney failure and EF <40% in adult patients are considered a poor prognostic sign for a kidney transplant and are turned down for kidney transplant. The prognosis of paediatric patients with kidney failure and cardiac dysfunction is not well known.

2.0 Objectives

2.1 General objectives

The primary objective of this study is to evaluate the effect of echocardiographic abnormalities pre-transplant on the post-transplant outcome. We will also determine the magnitude of significant cardiac abnormalities in children with kidney failure pre-transplant.

2.2 Specific objectives

2.2.1. To determine whether there is improvement in cardiac function on echocardiography post-transplant versus pre-transplant by comparing ejection fraction and/or fractional shortening.

2.2.2. To assess the structural changes (improvement or worsening) post-transplant compared to pre-transplant.

2.2.3. To assess how the CKD-associated pre-transplant factors affect the outcome of post-transplant echocardiography.

3.0 Methods

3.1. Study site

Red Cross War Memorial Children's Hospital (RCWMCH) is one of the dedicated children's hospital in Sub-Saharan Africa. The kidney unit provides tertiary care for children with kidney disease including chronic dialysis and transplantation. The unit is part of a sub-specialty pediatric ward of which twelve of the beds are allocated for kidney patients (including kidney transplantation patients) and includes dialysis facilities. The kidney unit admits between 30 and 40 patients per month (400/year). The unit is one of the three paediatric kidney transplant centers in South Africa and performs transplants in children as small as 10kg with paediatric and transplant surgeons. On average 10 kidney transplantations are done per year.

3.2 Study design

This is a single-center-based retrospective longitudinal descriptive and analytical study using charts and other medical record reviews.

3.3 Study population

3.3.1. Source population

All patients who underwent kidney transplantation at RCWMCH within the last 5 years from 1st of January 2018 to 31st December 2022.

3.3.2. Study population and sample size

All children who had kidney transplantation at RCWMCH during the study period

3.4 Eligibility criterion

3.4.1. Inclusion criteria

All forty-three patients who underwent kidney transplantation during the study period was included in the study.

3.4.2 Exclusion criteria

- Children who had congenital cardiac disease.
- Children with acquired heart disease due to other causes.

3.5. Study variable

3.5.1 Independent variable

- Control of hypertension pre-transplantation
- Dyslipidemia pre-transplantation
- Calcium and Phosphate levels
- Parathyroid hormone (PTH) level
- Hemoglobin before transplantation
- Duration of dialysis pre-transplantation
- Modality of dialysis pre-transplantation
- Cause of CKD
- Living vs diseased donor

3.5.2 Dependent variables

- Left ventricular function post-kidney transplant
- Improvement of structural changes post-transplant

3.6 Data collection

A structured case report form (CRF) was used to collect sociodemographic and anthropometric data, pre- and post-transplantation echo findings, and relevant blood results from the patient chart by the principal investigator. Data was recorded on an Excel spreadsheet which will be password protected and only the principal investigator (PI) will have access to it.

3.7 Data analysis and interpretations

Data analysis was done using the Statistical Package for Social Sciences version 27.0 (SPSS). A test for normality was done and a median value was used to correlate independent and dependent values. Wilcoxon signed-rank test was used to compare pre-and post-kidney transplant echocardiography findings for patients who had paired pre-and post-transplant echocardiography. The statistically significant association was be taken to be p values of <0.05.

3.8 Operational definitions

Cardiac abnormalities were defined as structural abnormalities or functional abnormalities noted on echocardiography.

3.9 Ethical approval

The ethical approval of this study was obtained from the Human Research Ethics Committee (HREC) at the University of Cape Town (UCT) and the Research Review Committee of RCWMCH (HREC REF 785/2023). Consent from the hospital CEO for data collection was also obtained.

4. Results

This retrospective study included 43 patients who underwent kidney transplantation over 5 years from 1st January 2018 to 31st December 2022 at Red Cross War Memorial Children's Hospital Cape Town South Africa 23/43(53%) were male and 20/43 were female (47%). 40/43 (93%) patients had their first transplant, while 3/43 (7%) patients had their second transplant during the study period. Most recipients were mixed race (60%) 26/43 followed by black 9/43(20 %) and white 8/43 (19%). Most were from the Western Cape 37/43 (86%) with only 6/43 (14%) from the Eastern Cape. The median age at transplant was 158 months. Almost half (51%) 22/43 of the recipients were blood group O followed by A (30%) 13/43, B (16%) 7/43, and AB (3%) 1/43.

In 14/43 (32%) cause of kidney failure was glomerular disease. Congenital Abnormalities of the kidney and Urinary tract (CAKUT) constituted 11/43(25%).3/43(7%) and 343(7%) patients had Haemolytic Uremic Syndrome (HUS. Of a total of 2(4%) metabolic illnesses, the patient had cystinosis and hyperoxaluria one each. Seven patients presented with kidney failure of unknown cause (16%). The three patients who had a second kidney transplant after a failed first transplant had renal dysplasia, Posterior urethral valve (PUV), and Cystinosis as a background disease. See Table

Sixty-three percent (27/43) had stunting before transplant. Thirty-seven (37%) of patients transplanted had severe stunting, 26% had moderate stunting and 37% had normal height for sex and age at transplantation. Regarding acute nutritional status, 28% had severe acute malnutrition(SAM) at transplant by body mass index (BMI) or Weight for height(WFH) Z score. Prevalence of SAM pre-transplant was reduced to 10% one year post-transplant. Obesity increased from 2 % to 13%.

The majority of the patients (84%) received Kidney transplants after a period of dialysis. While 16% of patients had pre-emptive kidney transplants. See Table 1. Fifty-three percent of patients received kidneys from diseased donors and 47% received them from living-related donors. 41/43 (95%) patients had isolated kidney transplants whereas, 2/43 (5%) had combined cadaveric kidney and liver transplants. The causes of kidney failure for combined kidney and liver transplant were Primary hyperoxaluria and autosomal polycystic kidney disease(ARPKD).Only 6/43 (14%)

patients required dialysis for delayed graft function while 86% had a good immediate function and did not require dialysis post-transplant.
Overall five patients died during the study period. First-year graft survival was 91% and 4/43 (9%) of patients died within the first year after transplant due to various reasons.3/43(7%) of the deaths had a glomerular cause of kidney failure. One had graft thrombosis immediately after transplant and died of COVID-19, the second also died of COVID-19 pneumonia and the third had a recurrence of the disease. The fourth patient with kidney failure due to primary hyperoxaluria underwent a combined kidney and liver transplant and died secondary to sepsis. One year of patient survival was equal to first graft survival (91%). The fifth death was a patient who initially presented with KF of unknown cause and died eighteen months after transplant after the patient presented with graft dysfunction.

The following parameters were used to define structural and functional abnormalities in echocardiography. Ejection fraction <55% and /Or Fractional shortening<28% was used to define systolic dysfunction. Left ventricular posterior wall thickness >11mm was used to define left ventricular hypertrophy. Mitral annular early flow velocity to late flow velocity (MV E/A) > 2.0 was used to define restrictive filling and diastolic dysfunction.
The majority of patients 32/43 (74%) had echocardiography before kidney transplantation, while (11/43)26% did not have echocardiography before transplant. Of the patients who had an echocardiographic evaluation, 17/42(54%) had normal echocardiographic findings while 15(46%) had abnormal echocardiographic findings. Four patients had clinical signs of heart failure requiring treatment pre-transplant (Figure 1).

Of thirty-two who had echoes, 15/32 (46.9 %%) had abnormal findings: 6/32 (19%) each with systolic and diastolic dysfunction respectively, and 3/32 (9%) with LV hypertrophy with no systolic or diastolic dysfunction (Figure 2).

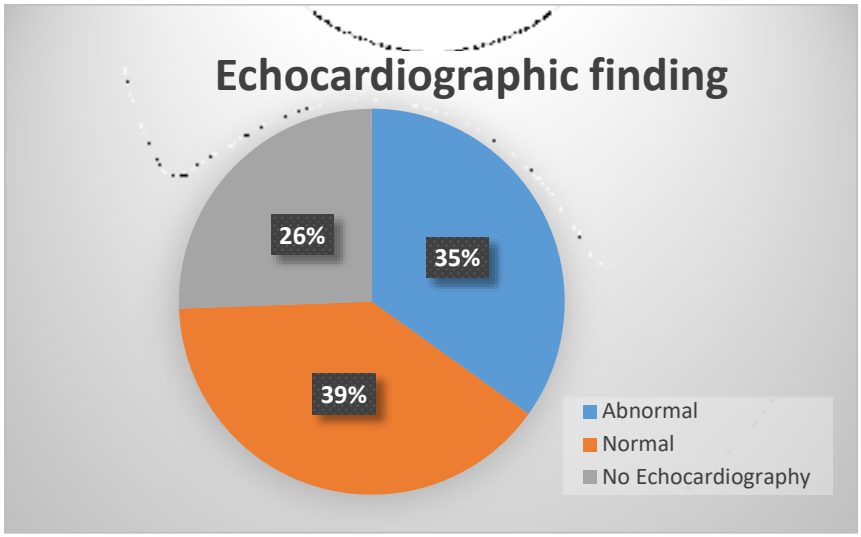

Figure 1 Finding of pre-transplant echocardiographic abnormalities (n=43)

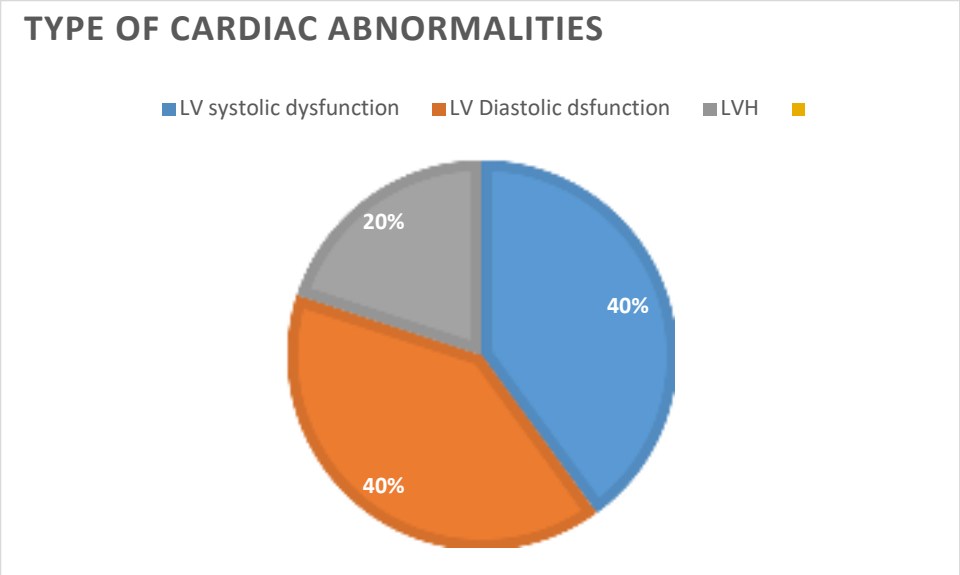

Figure 2 Type of pre-transplant echocardiographic abnormalities (n=15)

Variables	All patients n = 43
Age at transplant (months) median (IQR)	158.0 (106.0 – 179.0
Male sex	23 (5%)
CKD Cause	
Glomerular	14 (32.6%)
CAKUT	11 (25.6%)
Unknown	7 (16.3%)
HUS	3 (7.0%)
Failed the first transplant	3 (7.0%)
Cystinosis	1 (2.3%)
Hyperoxaluria	1 (2.3%)
Cystic kidney disease	3 (7.0%)
Dialysis pre-transplant	37 (86.0%)
Duration of dialysis pre-transplant (months) mean (SD)	16.1 (28.3)
Dialysis modality	
Pre-emptive	7 (16.3%)
PD	31 (72.1%)
HD	5 (11.6%)
Hypertension pre-transplant	35 (81.4%)
Nutritional status pre-transplant	
Severe acute malnutrition	4 (9.3%)
Normal	30 (69.8%)
Overweight/obese	5 (11.6%)

Stunting pre-transplant	
Severe	16 (37.2%)
Moderate	11 (25.6%)
Normal	16 (37.2%)
Anaemia pre-transplant	43 (100%)
Number of drugs used for anaemia	
0	1 (2.3%)
2	3 (7.0%)
3	39 (90.7%)
24-hour urine output (ml) *Mean (SD)*	522.3 (494.8)
Dipstick proteinuria median (IQR)	2.0 (1.8 – 3.0)
Quantitative proteinuria (UPCR) *median (IQR)*	0.5 (0.1 – 1.1)
Number of hypertensive drugs used pre-transplant median (IQR)	1.0 (1.0 – 2.0)
Target Ca Phosphate product grouped	
Target	14 (32.6%)
Increased risk	14 (32.6%)
High risk	15 (34.9%)
Lowest Haemoglobin before transplant *mean (SD)*	7.7 (1.8)
Highest PTH median (IQR)	52.8 (36.0 – 115.0)
Lowest Vitamin D level	
Deficient	8 (18.6%)
Insufficient	19 (44.2%)
Sufficient	16 (37.2%)
Number of drugs used for bone mineral disorders *mean (SD)*	2.1 (0.8)
Pre-transplant ejection fraction (n=32) *Mean (SD)*	59.7 (12.7)
Pre-transplant fractional shortening (n = 31) *Mean (SD)*	31.7 (8.4)
Donor type	
Living related	20 (46.5%)
Deceased	23 (53.5%)
Immediate graft function	
Excellent	37 (86.0%)
Delayed	6 (14.0%)
Inotropic support post-transplant	11 (25.6%)

Graft rejection	6 (14.0%)
Clinical heart failure	4
Nutritional status post-transplant (one year)	
Severe underweight	4 (9.3%)
Normal	30 (69.8%)
Overweight/obese	5 (11.6%)
Post-transplant ejection fraction (n=7) *Median (IQR)*	61.0 (45.0 – 74.0)
Post-transplant shortening fraction (n=7) *Median (IQR)*	32.0 (22.0 – 43.0)
Last, follow up estimated GFR *Mean (SD)*	59.4 (32.2)
Currently alive	38 (88.4%)

Table 1 dependent and independent variable
Five patients died during (11.6%). There were no significant differences in any variables between survivors and those who died.

There was no statistically significant difference between ejection fraction (EF) ($p = 0.5$) or shortening fraction (FS) ($p = 0.4$) pre- to post-transplant, in those who had paired samples (n=6) [using Wilcoxon signed rank test]. Similarly, there was no association between pre-transplant ejection fraction or shortening fraction and immediate graft functioning or graft rejection ($p > 0.1$) (Figure 3).

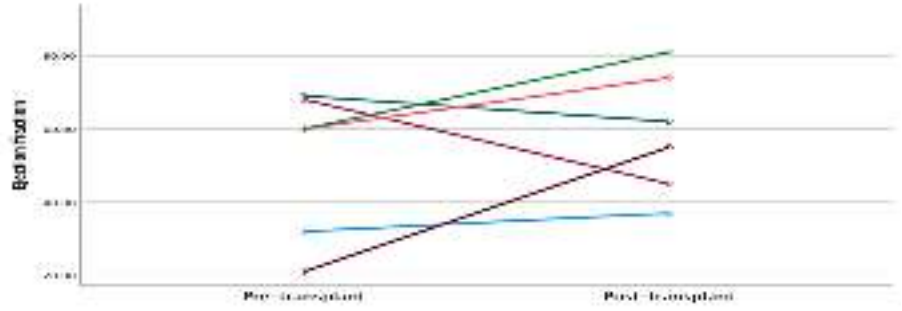

Figure 3 Change in ejection fraction % pre and post-kidney transplant

The graph shows the change in ejection fraction (EF) pre- to post- kidney transplant in those who had paired measures (n=6) – you can see that 4/6 improved, and two appeared to deteriorate. Both patients whose EF decreased from pre-transplant echocardiography are alive and doing well. There was no patient requiring heart failure treatment post-kidney transplant.

5. Discussion

In this descriptive study, we found about 46.9% of children who had echocardiographic screening pre-transplant had abnormal findings. This underlines that patients with CKD especially dialysis requiring KF need cardiac evaluation and echocardiography. The prevalence of diastolic and systolic dysfunction was equal among the pre-transplant echocardiographic findings. This is consistent with the finding that diastolic dysfunctions tend to occur before systolic dysfunction in children (6) (7). This could be explained by impaired LV relaxation in patients with kidney failure related to the high prevalence of hypertension, anaemia, high PTH, and proteinuria among study subjects.

In most studies left ventricular mass index, which is obtained by dividing calculated left ventricular mass for $height^{2.7}(g/m^{2.7})$ with cut of $>51 g/m^{2.7}$, was used for diagnosis of left ventricular hypertrophy. In our study left ventricular wall thickness was used as a parameter for the diagnosis of LVH. Left ventricular hypertrophy was reported to be high in children with kidney failure on maintenance dialysis (19). LVH appeared to be a less common finding in this study. This could be because most patients were on PD which has better volume control and less associated with LVH compared to LVH. Previous studies showed LVH to be the most common echocardiographic abnormality, this was not seen in this study(5).

This study also showed among patients who had paired pre and post-transplant echocardiography the EF improved in 4 patients. In 2 patients out of the 6, there was a decrement of EF. No patient had clinical heart failure post-kidney transplant. The echocardiographic studies post-transplant were done between 2 to 80 days post-transplant and may be too early to conclude improvement of echocardiographic abnormalities. In studies by Ashwin et al. among 6 patients who had cardiac dysfunction pre-transplant there was an improvement in ejection in all patients at one year post-transplant(15).

This study showed first-year graft survival was 91% (39/43). This was comparable to reports from Australian and New Zealand dialysis and transplant registry showed 93% one-year graft survival(20). This study showed there was no significant difference among patients who died and survived in any of the independent variables. This result shows at least a good short-term outcome for patients who had transplants and is very encouraging because children who remain on dialysis will have worsening cardiac function while children who were transplanted despite cardiac abnormalities showed improvement in LV systolic function.

6. Limitation

The limitation of this study is the small sample size and retrospective nature of the study.Pre-transplant echocardiography was not found for all patients. Even fewer echocardiography reports were found post-transplant. Inter-user variability about echocardiographic finding about echocardiographic findings is also possible.

7. Conclusions and recommendations

In this retrospective study, we were able to show the high prevalence of cardiac abnormalities among children undergoing kidney transplantation. Based on the kidney disease outcome quality initiative- KDOQI recommendations echocardiographic screening for dialysis requiring CKD should be performed within 3 months of dialysis initiation once dry weight has been achieved(21). The subsequent follow up based on individual patients. Patients with abnormal echocardiography should be evaluated with serial echocardiography post-transplantation. Echocardiography should also be performed for non-dialysis requiring CKD patients with hypertension to diagnose cardiac abnormalities early. The echocardiographic report should include major structural and functional findings pertinent to CKD patients.

We recommend prospective study with wider parameter evaluation such as left ventricular mass index (LVMI), 3 dimensional methods to determine EF and FS, evaluation of vascular changes such as carotid intima-media thickness (CMIT), pulse wave velocity (PWV), and additional evaluations such as 12 lead electrocardiography.

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