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Changes in Cardiac Parameters of Renal Allograft Recipients: A Compilation of Clinical, Laboratory, and Echocardiographic Observations

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ABSTRACT

Introduction. This study was undertaken to observe changes in cardiac parameters along with clinical and laboratory changes after renal transplantation.

Patients and Methods. Cardiac parameters were evaluated by M-mode 2-dimensional echocardiography before transplantation and at monthly intervals. All subjects had functioning grafts at the time of the evaluations.

Results. Fifty-two allograft recipients underwent pretransplant parameters for comparison to those at posttransplant months 1, 3, 6, and 12. When changes at month 1 and 3 were observed among 22 patients, improvements were evident at month 3. Comparisons of pretransplant versus month 3 showed systolic blood pressure (SBP), 161 ± 16 to 133 ± 26 mmHg ($P < .002$); diastolic BP (DBP), 101 ± 9 to 86 ± 11 mmHg, ($P < .006$); hemoglobin (Hgb), 7.3 ± 1.6 to 11.2 ± 3.9 g/dL ($P < .006$); left atrial diameter (LAD), 41 ± 5 to 35 ± 3 mm ($P < .001$); left ventricular muscle mass index (LVMI), 379 ± 114 to 248 ± 58 g/m^2 ($P < .001$); and left ventricular end diastolic volume index (LVEDVI), 96 ± 28 to 64 ± 17 mL/m^2 ($P < .002$). When changes at months 3, 6, and 12 were observed among 30 patients, improvements evident at month 3 were maintained. Comparisons of pretransplant and 3 and 12 months observations showed SBP, 157 ± 17, 131 ± 14, to 126 ± 10 mm Hg ($P < .001$); DBP, 97 ± 10, 83 ± 16, to 85 ± 6 mmHg ($P < .001$); Hgb, 7 ± 1, 13 ± 2, to 13 ± 2 g/dL ($P < .001$); LAD, 39 ± 7, 35 ± 3, to 34 ± 4 mm ($P < .05$); LVMI, 275 ± 91, 191 ± 38, to 159 ± 26 g/m^2 ($P < .001$); and LVEDVI, 87 ± 29, 56 ± 34, to 49 ± 24 mL/m^2 ($P < .001$).

Conclusion. Significant improvements in cardiac parameters were evident by the third month post-renal transplantation; the changes were maintained over a longer period among patients with functional grafts.

MORBIDITIES RELATED TO the cardiovascular system (CVS) are responsible for >90% of deaths in chronic kidney disease (CKD), even before reaching end-stage renal failure (ESRD).[1] Successful renal transplantation is followed by improvements in cardiac function and morphology. Significant reductions in left ventricular (LV) mass and normalization of cardiac output (CO) may be detected as early as 1 month posttransplant.[2] In this paper we compiled results from 2 studies of cardiac changes among kidney recipients in the early phase (first 3 months) and at the later part (up to 12 months) posttransplantation.

MATERIALS AND METHODS

In one study (group 1), 22 renal allograft recipients were investigated for alterations of selected cardiac parameters at 0 (immediate pretransplant), and 1 and 3 months after transplantation (group 1).[3] In the other study (group 2) 30 recipients were evaluated at 0, 3, 6, and 12 months posttransplant (group 2).[4] M-mode 2-dimensional, echocardiography together with relevant clinical and laboratory evaluations were performed at these intervals. The primary disease in the majority of patients was glomerulonephritis. All subjects received living-related donor kidneys and the grafts were

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0041-1345/08/$–see front matter
doi:10.1016/j.transproceed.2008.07.099

functioning at the time of evaluation. Immunosuppression included prednisolone, azathioprine, and cyclosporine.

Echocardiographic measurements were performed according to guidelines set by the American Society of Echocardiography,[5] including left atrial diameter in diastole (LADd); left ventricular internal diameter in diastole (LVIDd) and in systole (LVIDs); left ventricular posterior wall thickness (LVPWT); interventricular septal thickness (IVST); fractional shortening (FS%); left ventricular muscle mass index (LVMI); left ventricular end-diastolic volume index (LVEDVI); and vena caval diameter index (VCDI). Left ventricular hypertrophy (LVH) was defined when LVMI exceeded 131 g/m^2 in men and 100 g/m^2 in women. Concentric hypertrophy meant LVH with normal cavity volume and eccentric hypertrophy with dilation (cavity volume >90 mL/m^2).[6] Systolic dysfunction was diagnosed when the FS% was ≤25%. We also performed additional measurements of blood pressure (BP; systolic BP and diastolic BP), hemoglobin (Hgb) and serum creatinine (SCr) etc.

Results are expressed as mean values ± SDs. Analysis of variance (ANOVA), Student's t test and χ^2 test were used as appropriate as well as Pearson's correlation association. Values of $P < .05$ were taken as significant.

RESULTS

The age of 22 group 1 subjects was 31 ± 9 years with a dialysis duration of 5 ± 1.2 months. Compared with month 0, significant changes became evident at month 3, including BP (SBP, 161 ± 16 to 133 ± 26 mmHg [$P < .002$] and DBP, 101 ± 9 to 86 ± 11 mmHg [$P < .006$]); antihypertensive therapy (3 ± 1 to 2 ± 1 drugs per patient; $P < .001$); Hgb level (7.3 ± 1.6 to 11.2 ± 3.9 g/dL; $P < .006$); LADd (41 ± 5 to 35 ± 3 mm; $P < .001$); LVIDd (54 ± 6 to 47 ± 6 mm; $P < .02$); LVIDs 37 ± 7 to 31 ± 5 mm; $P < .05$); LVMI (379 ± 114 to 248 ± 58 g/m^2; $P < .001$); and LVEDVI (96 ± 28 to 64 ± 17 mL/m^2; $P < .002$). Other parameters like IVST, LVPWT, FS%, and VCDI did not show any significant change. At 3 months, 47% subjects were hypertensive. Pretransplant echocardiograms showed LVH in 100% of subjects, LV dilation in 52%, and systolic dysfunction in 18%. Follow-up evaluation showed LVH still present in all, and LV dilation had been reduced to 11%.

Group 2 subjects had a mean age of 31 ± 8 years and a dialysis duration of 7 ± 3 months. Compared with the 0 month values, significant changes had become evident at month 3 and persisted similarly at 6 and 12 months, including SBP (157 ± 17, 131 ± 14, and 126 ± 9 to 126 ± 10 mmHg; $P < .001$); DBP (97 ± 10, 83 ± 16, and 86 ± 8 to 85 ± 6 mmHg; $P < .001$); antihypertensive therapy (3 ± 1, 2 ± 1, and 2 ± 1 to 2 ± 1 drugs per patient; $P < .001$); Hgb (7 ± 1, 13 ± 2, and 13 ± 2 to 13 ± 2 g/dL; $P < .001$); LADd (39 ± 7, 35 ± 3, and 35 ± 3 to 34 ± 4 mm; $P < .05$); LVMI (275 ± 91, 191 ± 38, and 173 ± 39 to 159 ± 26 g/m^2; $P < .001$); LVEDVI (87 ± 29, 56 ± 34, and 63 ± 14 to 49 ± 24 mL/m^2; $P < .001$); VCDI (30 ± 7, 33 ± 3, and 34 ± 3, to 34 ± 2 mm/m^2; $P < .005$); and FS% (30 ± 7, 33 ± 3, and 34 ± 3 to 34 ± 2; $P < .05$). All subjects were initially hypertensive but by month 12, BP was within normal limit in 98% and 12% required no antihypertensive therapy. Pretransplant echocardiography showed LVH in 100% of patients with 50% of the concentric variety and systolic dysfunction in 30%. After transplantation, 100% showed significant reductions in LV mass by month 3. By months 12, LVEDVI and VCDI became normal in all and LVMI in 23%.

DISCUSSION

Significant improvements in cardiac parameters occur after renal transplantation. The alterations are evident in the early posttransplant period and continued over time, depending on BP control and renal function status.[7] The most prevalent cardiovascular abnormalities are LVH (up to 93%), systolic dysfunction (30%–60%), LV dilatation (27%), and diastolic dysfunction (17%).[8] Concomitantly, hypertension may persist in 45%–76% of subjects.[9] Among group 1 patients, BP was reduced in 47% at 3 months and in group 2. BP came to the normal range by 12 months among 98%. Changes in LV volume and mass were evident at 3 months in both groups with no further changes during the first year in group 2. This phenomenon can be explained principally by decreased ventricular diameter resulting from improvements in volume overload status as seen by the reduction of VCDI, but not LVPWT or IVST. Ferreira et al[8] observed similar reductions in interventricular diameters followed by reductions in muscle mass showing improvement in initial cardiac status. Additionally, the reductions in blood pressure with correction of anemia and decreased creatinine levels also influenced the improvements in LV parameters, as further substantiated by correlation studies (not shown) where increased hemoglobin and low serum creatinine negatively correlated with abnormal cardiac factors.[10]

We concluded that significant beneficial changes in cardiac function and morphology become evident by 3 months posttransplantation. The alterations, mainly due to reduction in diameters, are further influenced by correction of anemia and BP control. These changes are maintained over longer periods among subjects with functioning allografts.

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