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REGRESSION OF LEFT VENTRICULAR HYPERTROPHY AFTER SUCCESSFUL RENAL TRANSPLANTATION AMONG UREMIC PATIENTS

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ABSTRACT

Recognition of the natural history and responsible leading factors for regression of left ventricular hypertrophy after successful renal transplantation are very important. The aim of this study was to assess the regression of left ventricular hypertrophy after successful renal transplantation among uremic patients.In this study 27 uremic patients (18 males and 9 females) with an average age of 38.5 years were randomly selected. Left ventricular mass index (LVMI) was calculated before and after renal transplantation at the beginning, and at 4, 6 and 8 months.

The means of LVMI before and after transplantation were 180±19.3 g/m^2 and 133.8±16.8 g/m^2 respectively (p<0.001). The means of regression after transplantation at 0, 4, 6 and 8 months of follow up were 191g/m^2, 157.3 g/m^2, 147.8 g/m^2 and 138.8 g/m^2 respectively. There was a significant difference between the means of hemoglobin concentration and blood pressure before and after transplantation (p<0.001). For instance the means of hemoglobin concentration and blood pressure was 7.2±0.4 and 13.1±0.7 g/dL, 154±6 / 97±4.4, and 135±6.3/ 89±3.8 mmHg respectively.

This study showed that maximum left ventricular hypertrophy regression occurred 4 months after transplantation, then decreased to a minimum level of 147.8 and 135.8 g/m^2 at 6 and 8 months after transplantation respectively. Restoration of hemoglobin concentration and blood pressure to normal levels may be recognized as a main cause of left ventricular hypertrophy regression.

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INTRODUCTION

Over forty-five percent of deaths among patients with end stage renal disease result from cardiovascular events.[1-3] Cardiovascular complications present with increasing frequency after improving the survival of patients with end-stage renal diseases. It is estimated that about 26% of uremic patients have coronary artery disease and about 10% die due to myocardial infarction. The most common cause of mortality in end stage renal disease is left ventricular failure due to long-standing hypertension and anemia with left ventricular hypertrophy.[3,4] Left ventricular hypertrophy is occasionally complicated by sudden cardiac death due to ischemia or ventricular arrythmia without epicardial coronary artery disease. Systemic arterial hypertension occurs in 80-90% of patients at the beginning of hemodialysis.[4,5] Echocardiographic evidence of left ventricular hypertrophy due to systemic arterial hypertension and anemia has been

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shown in over 50% of patients with end-stage renal disease. The progression of left ventricular hypertrophy in uremic patients is less related to the level of blood pressure rather than anemia, since the process progresses regardless of good control of blood pressure and volume status. Many studies have shown a close correlation between amount of hemoglobin and left ventricular mass index.[3,6-10] Chronic volume retention, arteriovenous fistula, secondary hyperparathyroidism, increased level of catecholamines, and interstitial myocardial fibrosis due to uremia have a less important effect on this process.[2] Renal transplantation is the treatment of choice for end-stage renal disease. Successful renal transplantation is defined as a patient who has a creatinine level less than 2mg/dL after transplantation.[11-13] The aim of this study was to determine the regression of left ventricular hypertrophy after successful renal transplantation among uremic patients.

METHODS

This randomized clinical trial study was performed on 27 patients with end-stage renal disease referred to Golestan Hospital-Ahwaz from July 1998 to July 2000. All patients were under regular hemodialysis three times a week for 4 hours and were treated with supplemental calcium, iron and vitamin D, antihypertensive drugs such as atenolol, captopril, and niedipine.For some patients with severe anemia erythropoietin was administered. These patients were scheduled for renal transplantation. Renal, gastrointestinal, pulmonary, cardiac (including physical examination, electrocardiogram, chest x-ray, echocardiography and other invasive or noninvasive tests when appropriate), psychological, tissue compatibility and hematological examination and studies were done for the patients.The left ventricular mass index was calculated before and after renal transplantation. The echocardiographic instrument Hewlett-Packard model Sonos 1000 with transducer 2.5-3.5 MHZ was employed. The echocardiographic formula for calculation of LV mass index was 1.04 [(LVID + IVSD + PWT) - (LVIDd)] - 13.6 / BSA at parasternal, apical, two, four and five standard chamber view (m-mode, two dimensional, color Doppler).[1] Standard triple immunosuppressive therapy after transplantation including cyclosporine, prednisolone, and azathioprine plus antibiotics and antihypertensive medication was initiated. All cases received antihypertensive treatment for 4 months after transplantation and then only 50% took these drugs. Hemoglobin concentration returned to normal within 2 months after transplantation. The LVMI was calculated during the 8 months after renal transplantation. All patients were under close supervision of one nephrologist and one cardiologist during the study. The confidence level of LVMI was assessed as 95 percent. The mean of LVMI and confidence level was calculated by $\alpha<0.05$, and the regression rate of hypertrophy was estimated according to the formula: mean of LVMI before RTX-mean of LVMI after RTX/mean of LVMI before RTX×100.

RESULTS

Subjects were 27 uremic patients with a male/female ratio of 2/1 with a mean age of 38.5 years. All cases were in good condition and had creatinine levels of less than 2 mg/dL after renal transplantation. 88% of patients (24 cases) had left ventricular hypertrophy (82.6% for males and 100% for females). Left ventricular systolic dysfunction was observed in 20% of patients (6 cases) with a higher incidence rate for males. Left ventricular systolic dysfunction improved in all cases within 8 months after transplantation. The pattern of left ventricular hypertrophy was eccentric in 70%, concentric in 25% and asymmetric in 5% of patients. The echocardiographic indices in 27 patients before and after transplantation are shown in Table I. The means of the LVMI before and 8 months after transplantation were 180.1±19.3gr/m^2 and 133.8±16.8 g/m^2. The difference between the two means was statistically significant ($p<0.001$). The females had 161.6±17.8 and 125.5±26.8 g/m^2 and males had 189.4±26.8 and 137.9±21.7g/m^2 LVMI means before and after transplantation. There was no significant statistical difference between males and females.The hemoglobin concentration was 7.2±0.4g/dL and 13.1±0.7g/dL before and after transplantation with 45% increase in this level ($p<0.001$). The blood pressure was 154±4.6/97.4±4.4 and 135±6.4/89.6±3.9 mmHg before and after transplantation. The decline in systolic pressure was statistically significant ($p<0.001$). The study of echocardiographic indices of left ventricular function before and after transplantation showed significant changes. Left ventricular function indices such as end systolic, diastolic, internal dimension and fraction of shortening showed statistically significant changes ($p<0.05$). The means of LVMI were 191, 157.9, 141.8, and 135.8g/m^2 at 0, 4, 6 and 8 months respectively. This was estimated to be about 17% at 0-4, 10% at 4-6 and 4% at 6-8 months after successful transplantation.

DISCUSSION

This study showed no ideal left ventricular mass among all cases after 8 months of renal transplantation. Regression process was followed up during 8 months by echocardiographic examination exclusively but resolution of interstitial fibrosis due to uremia should be noted.[14-17] Many investigators believed that virtual regression of the hypertrophy depends on resolution of fibrosis.[18-19] However, this study did not show a complete resolution of hypertrophy. Recipients of renal transplantations have a higher incidence of cardiovascular events than the general population.[20] .Consequently, a large amount of decrease in left ventricular mass with improving function is associated with a lower risk

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Table I. Echocardiographic indices in 27 patients before and after renal transplantation.

	Before	SD	After	SD	P-Value
Systolic BP	154.6	17.42	135.1	16.87	<0.001
Diastolic BP	97.4	11.55	89.7	10.27	<0.02
Hemoglobin	7.2	1.05	12.9	1.84	<0.001
Creatinine	7.5	2.43	1.2	0.35	<0.001
LVMI	180.2	51.04	133.8	44.63	<0.001
End SV	60.5	27.90	43.3	16.92	<0.05
End DV	101.5	42.43	83.6	28.71	<0.05
LVPW	1.2	0.25	1.1	0.18	<0.1
IVSD	1.3	0.62	1.1	0.24	<0.02
LVID	5.5	0.25	4.9	0.57	<0.001
FS	37	8.76	34.1	8.49	<0.02

BP=Blood pressure. Hemoglobin= Hemoglobin concentration. Creatinine= Creatinine level. LVMI=Left ventricular mass index. End SV=End systolic volume. End DV= End diastolic volume.LVPW=Left ventricular posterior wall thickness. IVSD=Interventricular septal thickness in diastole. LVID= Left ventricular internal dimension. FS= Fraction of shortening. SD= Standard deviation.

of cardiovascular events.[21] Function studies by echocardiography have shown significant statistical changes in end systolic, end diastolic volume, internal dimension and fraction of shortening after transplantation. This study showed a nonsignificant difference in the regression process by sex. This is not reinforced by other experimental studies which have taken place among animals.[22] Many studies have shown a close correlation between left ventricular hypertrophy regression and anemia after renal transplantation.[23] We evaluated the relationship between left ventricular hypertrophy regression with anemia and hypertension after renal transplantation. We observed that the left ventricular hypertrophy regression had a close relationship with return of hemoglobin concentration to normal. The left ventricular hypertrophy regression was not completed after treatment with human erythropoietin in some studies.[2,6,14,15,23] Others have shown a close relationship between left ventricular hypertrophy regression and pulse pressure.[8,24-25] Improvement of anemia and better control of hypertension probably have a main role in the regression process. Systolic compared to diastolic blood pressure may have a closer relationship with left ventricular hypertrophy regression.[26] We found that anemia and systolic blood pressure have important roles in left ventricular hypertrophy, so it should be a very important factor in improving the regression process.[27] In this study the maximal range of left ventricular hypertrophy regression was obtained during 4 months after transplantation and then the progress was slow. The regression statistical analysis showed nonuniformity in its process. Other studies have shown a peak of regression at 2 years after transplantation and this process persisted for 3 to 4 years.[28] Although this nonuniformity is probably due to the role of medication and younger age of our patients. The glucocorticoids, immunosuppressive and regulator drugs (for example cyclosporine) had a tendency in inducing and aggravating hypertension after transplantation.[29] Recently in renal transplant recipients the AT1 receptor antagonist losartan has been shown to reduce left ventricular hypertrophy without altering systolic or diastolic left ventricular function.[30] We believe that with better control of blood pressure and anemia, a shortened interval between hemodialysis and transplantation, and the advent of new drugs with lower complications will decrease the rate of left ventricular hypertrophy as an independent risk factor for these patients.

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