Significant Regression of Left Ventricle Hypertrophy and Dilatation One Year After Kidney Transplantation

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Introduction and Background

Cardiovascular disease (CVD) is a major cause of morbidity and mortality in dialysis patients and Kidney transplant recipients (KTRs). Such patients may have left ventricular dilatation (LVD) and/or hypertrophy (LVH), which represent a significant risk factor for adverse cardiovascular outcomes (1).

Risk factors include age, diabetes, and other traditional cardiovascular risk factors, in addition to several other 'non-traditional' risk factors including anemia, abnormalities of calcium phosphate metabolism, lipid abnormalities, altered homeostatic factors, inflammation, oxidative stress, reduced bioavailability of nitric oxide and autonomic dysfunction (2).

In patients on hemodialysis, LVH is an independent risk factor for cardiovascular death. Pressure overload, resulting from hypertension and related to arterial stiffness and vascular calcification leads predominantly to concentric LVH. Anemia and volume overload could be assumed to cause left ventricular dilatation, due to salt and water overload (3).

Approximately 50% of KTRs die with a functioning transplant, with approximately 50% of these deaths from CVD or stroke (4).

Results

After 1 year, KTRs showed significant reduction of left ventricle end diastolic and systolic dimensions from 5.7 ± 0.2 to 5.6 ± 0.1 cm ($p < 0.05$) and from 3.8 ± 0.4 to 3.7 ± 0.1 cm ($p < 0.05$), respectively.

Also, there was a highly significant regression of left ventricle mass (LVM) from 298 ± 30 to 226.5 ± 54 gm ($p < 0.001$). On the other hand, patients who continued haemodialysis, showed no significant regression. There was insignificant regression of CIMT in both dialysis patients and KTRs

Methods

This study included 2 groups of patients (25 each) who had been on regular haemodialysis for a period of 12-18 months. Group 1 continued on haemodialysis, while kidney transplantation (KTx) was done for group 2.

Evaluation of the cardiac status was done at the start of the study and after 1 year later; clinically and using echocardiography and Doppler study of carotid intima-media thickness (CIMT).

All patients had haemodialysis done using an arteriovenous fistula or graft. Evaluation at the start of the study and at the end included clinical assessment of the cardiovascular symptoms in the form of chest pain, dyspnoea, transient ischemic attacks, intermittent claudication, and symptoms of anaemia. Laboratory workup was done (e.g. haemoglobin level and electrolytes), echocardiographic assessment of the left ventricular mass and carotid duplex examination of the carotid intima-media thickness.

Clinical evaluation, laboratory and imaging workup was done again at the end of the study for follow up and comparison between the two groups.

Conclusion

Successful kidney transplantation led to a significant regression of left ventricular hypertrophy and dilatation in patients on regular hemodialysis treatment. However, transplantation did not lead to a significant regression of CIMT. This could be attributed to the severity of already established vascular lesions (including calcification) in CKD patients on regular hemodialysis, even after 1 year of a successful kidney transplantation.

References

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