Hypertension following Kidney Transplantation

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High blood pressure and its consequence continue to be a vexed problem to the nephrology teams; in their patients of End Stage Kidney Failure who are being rehabilitated by kidney transplantation surgery. It continues to affect 50-80% of such patients.1

Relationship between poor control of blood pressure and reduced allograft survival have been clearly demonstrated and is similar to the effect of uncontrolled hypertension on progression of chronic kidney disease to stage V needing dialysis.2 A study done by Kaul and Sharma et al in this issue of the journal; has shown that age more than 40 years male, sex, use of Calcineurine Inhibitor (Cyclosporine) and use of high dose of steroids were significantly associated with post transplant hypertension in multivariate analysis.

Given the association between hypertension and deterioration in allograft outcome one would believe that active antihypertensive therapy would improve graft prognosis. It has been proposed that intraglomerular hypertension with proteinuria, secondary both to inadequate nephron mass and loss of functioning nephrons may contribute to chronic allograft failure. In cyclosporine treated subjects; it is associated with sodium retention and renin-angiotensin system suppression and hence a relative lack of renoprotective effect of ACE inhibitor / A1 Receptor Antagonist in them. These two factors will need different management strategies in the use of antihypertensive medication in individual patients; if one has two improve long term progression in grafted patients.3 There are several studies available to show that both systolic and diastolic blood pressure need to be effectively controlled to improve the prognosis of such grafted individuals; as shown in the current published study from SGPGI Lucknow.

The extent of renal injury associated with chronic allograft dysfunction, resulting from immunological damage from chronic rejection vs. nonimmunologically mediated haemodynamic factor is still debated.4 Both immunological and nonimmunological mediated effect of chronic rejection are further compounded by vasoconstrictive action of cyclosporine; predominantly on afferent arteriole. Taking these in to account; Calcium Channel Blockers and Converting Enzyme Inhibitor/A1 Receptor; both independently and combined have been used; but there are no controlled long term studies which have reached any specific conclusion.

Hence several new strategies for specific enhancement of glomerular perfusion are on the horizon.5 These include endothelin receptor antagonist; neutral endopeptidase inhibitor such as conodoxatril. However long term prospective studies are needed to assess their efficiency on chronic allograft outcome.

As mentioned in the current article; correctable risk factors like renal artery stenosis with renal angioplasty, and bilateral nephrectomies of native kidney in a few selected cases; not responding to multiple antihypertensive drugs; need specific study in a given individuals and attention to be paid to corrective therapy to help improve long term allograft prognosis.

Research is also needed to develop specific diagnostic criteria for assessment of role of extent both immunological factors in individual patient's. These would enhance our understanding in the management strategies in such a patients.

References