

Managing Hypertension in Post-Renal Transplant Recipients

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Abstract

Post-transplant hypertension is caused by a donor, recipient and immunosuppression related factors. Optimal control of blood pressure in renal transplant recipients is necessary for long-term allograft and patient survival.

Keywords: Hypertension; Post-Renal Transplant

Introduction

Hypertension is highly prevalent in individuals with chronic kidney disease (CKD) increasing from 36% in CKD stage 1 to 84% in CKD stages 4 and 5 [1] and frequently persists even in patients who receive kidney transplant. Uncontrolled hypertension in kidney transplant recipients is associated with shortened allograft as well as patient survival. Cardiovascular (CV) diseases in adult patients is the most common cause of death in renal transplant recipient and hypertension accounts for a major portion of this increased substantial CV risk.

Donor related factors for hypertension include positive family history, pre-existing donor hypertension, advanced donor age, prolong ischemia time and recipients of lesser quality. In addition to kidney disease, essential hypertension, diabetes and obesity, factors like saline loading, induction of high dose immunosuppression, post-transplant renal artery stenosis and antibody mediated rejection contribute to new onset hypertension in formerly normotensive renal transplant recipients. Hypertension is successfully managed in end stage renal disease but very less is known about optimal antihypertensive therapy in kidney transplant recipients. The selection of the drug should therefore depend on the patient's comorbid condition.

Calcium channel blockers (CCB) are used as antihypertensive agents in renal transplant recipients. It decreases the renal vasculature resistance and can counter the vasoconstrictive effect of calcineurin inhibitors (CNIs). Reimdsijk, *et al.* found that the patients on CCB post-transplant had better serum creatinine and blood pressure control at month 3 and 12th after the transplant as compared to the patients on other antihypertensive regimens [2]. However, other studies have shown that CCB, an ACE I and an alpha-blocker are equally effective as antihypertensive regimen [3]. Due to inhibition of CYP3A4, dihydropyridine calcium channel blockers (diltiazem, verapamil, and nifedipine) are not recommended in the transplant patients as they increase the tacrolimus and CsA levels [4].

Renin-Angiotensin-Aldosterone system (RAAS) Inhibition can slow the progression of CKD in diabetic and non-diabetic proteinuric patients [5,6]. The efficacy of RAAS inhibition in the kidney transplant recipients showed a clear benefit both in terms of graft or patient survival [7,8].

Thiazide Diuretics can be used as an effective treatment for hypertension as well as the beta-blocker.

Conclusion

For prolonged graft survival lower blood pressure ($\geq 130/80$ mmHg) should be the aim in kidney transplant recipients. Immediately after transplant, focus more on volume optimization with a loop or thiazide diuretics, beta blockers in coronary artery disease or arrhythmias and calcium channel blockers, thus improving kidney functions.

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