Medical Management of the Kidney Transplant Recipient

A Practical Approach for the Primary Care Provider

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KEYWORDS

• Kidney transplant recipient • Hypertension • Diabetes • Obesity • Dyslipidemia

KEY POINTS

- Kidney transplant recipients (KTR) commonly present with a multitude of metabolic derangements that have a major impact on long-term outcomes.
- The successful management of the KTR requires a collaborative effort between the primary care physician and the transplant center.
- Hypertension is present in more than 80% of these patients and often requires multiple medications to achieve the established, aggressive systolic and diastolic targets.
- Diabetes mellitus can be present before transplant or occur as a de novo medical issue in the posttransplant period. Corticosteroids and other immunosuppressives are contributory.
- Dyslipidemia and obesity are challenging problems commonly encountered in the medical management of the KTR and require effective treatment strategies on a long-term basis.

INTRODUCTION

Evolving trends in health care have placed the primary care physician (PCP) at the forefront of long-term patient management. Not infrequently, PCPs are required to manage and make clinical decisions with patients with complex medical conditions that are not in disciplines for which they have received formal training. An example of this is the kidney transplant recipient (KTR), a patient that commonly has multiple comorbidities, including hypertension (HTN), diabetes mellitus, dyslipidemia, and obesity. This is on a background of end-stage renal disease further complicated by the complexity of long-term maintenance immunosuppression with medications that

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present a host of adverse reactions and drug–drug interactions. With more than 17,000 kidney transplants being performed annually in the United States and almost 200,000 recipients alive with a functioning graft, it is not uncommon for PCPs to have several KTRs in their practice. The purpose of this review is to highlight medical management issues of practical usefulness to the PCP who is involved in the care of KTRs.

HYPERTENSION

Hypertension is often encountered in the management of KTRs with the prevalence reported to be from 50% to 90%.[1] Poorly controlled blood pressure has been shown to be an independent risk factor for cardiovascular disease (CVD) after kidney transplantation and is also associated with an increased risk of graft failure.[2–4] Studies in the general population have conclusively demonstrated that aggressive treatment of HTN is associated with a significant reduction in the incidence of cardiovascular events and a reduction in overall patient mortality.[5] These outcomes have been demonstrated in both observational and randomized clinical trials and although similar trials have not been performed in the transplant population, these results have been extrapolated to apply to the KTR as well.

The goals for treatment of HTN in the KTR have been mostly adopted from guidelines directed to the care of the general population. This includes reducing the systolic blood pressure to 140 mm Hg or less and diastolic blood pressure to 90 mm Hg or less for low-risk patients. In high-risk patients, such as those with diabetes mellitus (DM) or chronic kidney disease (CKD), the recommendation is to reduce the systolic blood pressure to 130 mm Hg or less and diastolic blood pressure to 80 mm Hg or less.[5,6]

The pathogenesis of HTN in the KTR shares many similar etiologies as in the general population; however, several other possibilities should also be considered. It has been clearly demonstrated that certain immunosuppressive medications can contribute to an increase in blood pressure or worsen blood pressure control. Calcineurin inhibitors (CNI) such as tacrolimus and cyclosporine are potent vasoconstrictors, and corticosteroids can be permissive of salt and water retention. Other clinical scenarios that might contribute to posttransplant HTN include acute and chronic allograft dysfunction and stenosis of the transplant renal artery. A careful assessment for each of these possibilities is part of the management of the KTR with poorly controlled or difficult to control blood pressure.

Treatment

It is generally accepted that all antihypertensive agents are useful in KTRs and the selection of the initial antihypertensive medication may be determined by factors such as race, the presence of comorbidities, and/or posttransplant complications (**Fig. 1**).

Calcium channel blockers are the most commonly prescribed antihypertensive for the KTR. The dihydropyridines, by virtue of their direct vasodilating properties, are a popular choice because they counteract the vasoconstrictive properties of the CNIs. Nondihydropyridine calcium channel blockers, such as verapamil and diltiazem, are less commonly used and should be avoided if at all possible because of their known inhibition of CYP3A with consequent increase in CNI blood levels.[7] Careful monitoring of CNI blood levels is warranted if calcium channel blockers, especially nondihydropyridines, are used.

β-Blockers are particularly useful in cases in which comorbidities such as congestive heart failure or coronary artery disease are present. The benefit of this class of antihypertensive on cardiac outcomes has been unequivocally demonstrated in the