

New Onset Diabetes Mellitus After Transplantation: Can We Prevent It?

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New-Onset Diabetes After Transplantation (NODAT) is an increasingly recognized severe metabolic complication of kidney transplantation causing lower graft function and survival and reduced long-term patients survival mainly due to cardiovascular events. Prior studies show that approximately 15–30% of nondiabetic kidney transplant recipients develop NODAT in the first year after transplant. Pretransplant oral glucose tolerance test (OGTT) also plays an important role in identifying prediabetes state such as impaired fasting glucose, which is associated with increased in the incidence of NODAT, therefore providing an opportunity to prevent the onset of NODAT, proposing a less diabetogenic immunosuppressive protocol after transplantation. The screening test to detect NODAT should be performed in the time frame between 1 and 12 months after transplantation. Risk factors for the development of NODAT are categorized as non-modifiable (e.g., advance age; recipient male gender; nonwhite ethnicity; family history of diabetes mellitus; HLA A30, B27 and B42; acute rejection history; and polycystic kidney disease), modifiable or potentially modifiable (e.g., obesity; hepatitis C virus and cytomegalovirus infection; physical inactivity; glucocorticoids, tacrolimus, cyclosporine and sirolimus; proteinuria; and hypomagnesemia). These risk factors reflect either inherited or acquired defects in insulin sensitivity and β cell function that contribute significantly to hyperglycemia. The best therapeutic approach should be started with prevention, trying to optimize modifiable risk factors, such as obesity and immunosuppressive therapy, and encouraging a lifestyle modification. Diet and regular exercise along with an appropriate nutrition therapy have shown to improve glucose metabolism and prevent the development of NODAT. Before kidney transplantation, successful treatment of hepatitis C can potentially reduce risk of NODAT. In addition, resting the β cell with basal insulin and optimizing β cell protection with tighter control to near-normoglycemic treatment could further reduce the number of patients with future impaired glucose tolerance and NODAT after transplantation. Transplant recipients who receive belatacept have been shown to be associated with a more favorable metabolic profile and a lower incidence of NODAT compared with those receive cyclosporines. Furthermore, there are ongoing clinical trials evaluating the efficacy and safety of treatment with sitagliptin, vildagliptin or pioglitazone in preventing the onset of NODAT. Once NODAT has been diagnosed, the antihyperglycemic agents have needed to achieve a tight glycemic control without impairing renal function.