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Meeting Report

Proceedings From an International Consensus Meeting on Posttransplantation Diabetes Mellitus: Recommendations and Future Directions

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A consensus meeting was held in Vienna on September 8–9, 2013, to discuss diagnostic and therapeutic challenges surrounding development of diabetes mellitus after transplantation. The International Expert Panel comprised 24 transplant nephrologists, surgeons, diabetologists and clinical scientists, which met with the aim to review previous guidelines in light of emerging clinical data and research. Recommendations from the consensus discussions are provided in this article. Although the meeting was kidney-centric, reflecting the expertise present, these recommendations are likely to be relevant to other solid organ transplant recipients. Our recommendations include: terminology revision from new-onset diabetes after transplantation to posttransplantation diabetes mellitus (PTDM), exclusion of transient posttransplant hyperglycemia from PTDM diagnosis, expansion of screening strategies (incorporating postprandial glucose and HbA1c) and opinion-based guidance regarding pharmacological therapy in light of recent clinical evidence. Future research in the field was discussed with the aim of establishing collaborative working groups to address unresolved questions. These recommendations are opinion-based and intended to serve as a template for planned guidelines update, based on systematic and graded literature review, on the diagnosis and management of PTDM.

Abbreviations: AGM, afternoon glucose monitoring; CNI, calcineurin inhibitor; GRADE, Grading of Recommendations Assessment, Development and Evaluation; NODAT, new-onset diabetes after transplantation; OGTT, oral glucose tolerance test; PTDM, posttransplantation diabetes mellitus

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Introduction

Previously published consensus guidelines on the diagnosis and management of diabetes mellitus after transplantation acknowledged the importance of posttransplant diabetes in all forms of solid organ transplantation and

the need for pro-active, multi-disciplinary management (1,2). As these were based on conferences held a decade ago, an International Expert Panel of clinicians/researchers was recently convened (Vienna, Austria, September 8–9, 2013) with two objectives: (1) update previous consensus statements and (2) debate current gaps in our clinical evidence base. The panel comprised 24 transplant clinicians, diabetologists and scientists with an active interest in the field. Invitations were based upon a meeting prerequisite to systematically review existing literature for presentation at an open scientific session, encouraging debate and discussion (3). This session contributed to the proceedings of the subsequent closed meeting of the International Expert Panel the following day. While the focus was on kidney transplantation, reflecting the published literature, the principles are likely relevant to all forms of solid organ transplantation.

This Meeting Report summarizes our major recommendations from the consensus meeting, with quality of evidence graded in line with GRADE (Grading of Recommendations Assessment, Development and Evaluation) definitions (4). GRADE provides a systematic approach to grade quality of evidence and strength of recommendations. Consensus opinion was to provide the following recommendations: high (Recommendation 4), moderate (Recommendations 2, 3, 5 and 6) and none possible (Recommendation 1 and 7). Readers requiring comprehensive literature reviews as background information are recommended recent publications in this area (5,6). It is anticipated these opinion-based recommendations will form the template for a planned comprehensive update to existing guidelines.

Recommendation 1: Change Terminology From New-Onset Diabetes After Transplantation Back to Posttransplantation Diabetes Mellitus (PTDM)

The term new-onset diabetes after transplantation (NODAT) was adopted to acknowledge the pathophysiological consequences of transplantation on glycemic metabolism. However, the term may be misleading, as diabetes is often unrecognized (7,8). The term NODAT implies exclusion of diabetes prior to transplantation, but effective pretransplant screening is impractical for many centers.

The term posttransplantation diabetes mellitus (PTDM) addresses these shortcomings by simply describing newly diagnosed diabetes mellitus in the posttransplantation setting (irrespective of timing or whether it was present but undetected prior to transplantation or not). The term PTDM should be utilized for clinically stable patients who have developed persistent posttransplantation hyperglycemia (see Table 1). The term prediabetes should be utilized for patients with posttransplantation hyperglycemia not reaching diagnostic thresholds for PTDM (impaired fasting glucose and/or impaired glucose tolerance) (Table 1). Fasting glucose has a low sensitivity for diagnosing PTDM, as kidney allograft recipients have relatively preserved fasting glucose concentrations after an oral glucose tolerance test (OGTT) (9–11). Consequently, lowering the threshold for impaired fasting glucose in the screening for PTDM seems appropriate and the American Diabetes Association cutoff (5.6 mmol/L [100 mg/dL]) was preferred over the World Health Organization cutoff (6.1 mmol/L [110 mg/dL]). These updated terms are utilized for the rest of this report.

Recommendation 2: Exclude Transient Posttransplantation Hyperglycemia From PTDM Diagnosis

Hyperglycemia is exceptionally common in the early posttransplant period, detectable in approximately 90% of kidney allograft recipients in the early few weeks (12,13). Hyperglycemia can also occur as a consequence of rejection therapy, infections and other critical conditions. While identifying transient posttransplantation hyperglycemia is important, being an important risk factor for subsequent PTDM (14), ubiquitously labeling the majority of kidney allograft recipients with PTDM in the immediate posttransplant setting is not helpful. A formal diagnosis of PTDM is best made when patients are stable on their likely maintenance immunosuppression, with stable kidney allograft function and in the absence of acute infections.

Recommendation 3: Expand Screening Tests for PTDM Using Postprandial Glucose Monitoring and HbA1c to Raise Suspicion, While Oral Glucose Tolerance Tests Remain the Most Important

The transplant community lacks data linking glycemic parameters with long-term macrovascular (e.g. myocardial infarct, stroke) and microvascular (e.g. retinopathy, nephropathy) complications, remaining dependent on outcome studies from the nontransplant population (15). As no glycemic indicator posttransplantation has demonstrated superiority with regard to long-term outcomes, the optimal measure remains unclear. At present, the OGTT is considered the gold standard for diagnosing PTDM. OGTTs identify more patients with diabetes posttransplantation than fasting glucose measurement alone (9–11), a similar observation to the general population, but detection is higher due to different pathophysiology between PTDM and type 2 diabetes (6,16). An OGTT also allows diagnosis of impaired glucose tolerance to be made, which is an independent risk factor for long-term development of PTDM, cardiovascular disease and mortality when tested either before (17) or after transplantation (18,19). However, OGTTs are not widely used as they are time consuming and impractical in a large transplant program.

Table 1: Diagnostic criteria for diabetes mellitus and prediabetes by the American Diabetes Association (ADA)

	ADA[1]
Diabetes mellitus	Symptoms of diabetes plus RPG $\geq$ 200 mg/dL (11.1 mmol/L) OR FPG $\geq$ 126 mg/dL (7.0 mmol/L) OR 2HPG $\geq$ 200 mg/dL (11.1 mmol/L) during an OGTT OR HbA1c $\geq$ 6.5%
Prediabetes	
Impaired fasting glucose	FPG 100–126 mg/dL (5.6–6.9 mmol/L)
Impaired glucose tolerance	FPG $<$ 7.0 mmol/L AND 2HPG 7.8–11.0 mmol/L
Increased risk of diabetes	HbA1c 5.7–6.4%
Normal glucose tolerance	FPG $<$ 110 mg/dL (5.6 mmol/L) AND 2HPG $<$ 140 mg/dL (7.8 mmol/L) AND HbA1c $<$ 5.7%

RPG, random plasma glucose; FPG, fasting plasma glucose; 2HPG, 2-h plasma glucose after an oral glucose.
[1]A confirmatory laboratory test based on measurements of venous plasma glucose must be done on any subsequent day in the absence of unequivocal hyperglycemia accompanied by acute metabolic decompensation. Symptoms of diabetes include polyuria, polydipsia and unexplained weight loss. Random plasma glucose is defined as any time of day without regard to time since last meal. Fasting is defined as no caloric intake for at least 8 h. The oral glucose tolerance test should be performed using a glucose load of 75 g anhydrous glucose dissolved in water.

HbA1c-based diagnosis is endorsed for diagnosis of diabetes mellitus in the general population (20) and we recommend elevated HbA1c be used to recognize PTDM (see Table 1). Caution must be exercised with its use early posttransplantation, as a normal HbA1c will not exclude diagnosis in the presence of posttransplantation anemia and/or dynamic renal allograft function (21). However, HbA1c 5.7–6.4% or higher in this early period would indicate the need to follow up with a recognized diagnostic test, although HbA1c greater than 6.5% is unlikely to be a false positive. Shabir et al (22) suggest optimum HbA1c cut-off values for predicting PTDM at 3 and 12 months of 44 mmol/mol (6.2%) and 48 mmol/mol (6.5%), respectively (latter equivalent to general population), but this analysis was based on a small cohort of 71 kidney allograft recipients and requires further validation.

Yates et al (23) recently reported afternoon glucose monitoring (AGM) in kidney allograft recipients, using capillary blood glucose, was more sensitive to both OGTT and HbA1c for detecting hyperglycemia in the initial 6-week posttransplant period in patients on corticosteroid containing regimens. However, this approach has not been validated for diagnosis of PTDM. Thus, testing for AGM in the early postoperative period may better identify individuals who should receive an OGTT or other recognized diagnostic testing.

The use of screening strategies should help to streamline diagnosis of PTDM, through identification of a subset of high-risk patients who should undergo further testing. One study demonstrated the benefit of a screening algorithm based upon fasting glucose and/or HbA1c, thereby reducing overall number of OGTTs required (9). The superiority of this streamlined approach over more widespread OGTT testing for PTDM remains to be determined.

Surveillance for glycemic abnormalities pretransplantation, including OGTT when possible, will help identify patients who have undiagnosed diabetes (8) or prediabetes (17). Studies have shown the utility of glucose-based diagnostic criteria pretransplantation (8), but using HbA1c is fraught with difficulty in patients with severe renal impairment or end-stage kidney disease. Kidney transplant candidates should have an annual check of glycemic status, either in the form of fasting glucose or risk-stratified OGTT (based upon center-specific screening algorithm). However, evidence is insufficient to recommend OGTTs for all kidney transplant candidates unless it forms part of a risk-stratified algorithm (8).

Recommendation 4: Identify Patients at Risk for PTDM

Risk factors for PTDM are well established (24), encompassing both general (e.g. age, family history of diabetes, prior history of glucose intolerance (25)) and transplant-specific (e.g. immunosuppression) factors, with accruing risk factors associated with greater PTDM risk. Novel targets continue to be identified, incorporating an improved understanding of metabolic syndrome and identification of select genetic polymorphisms as PTDM risk factors. Israni et al (26) found posttransplant metabolic syndrome independently associated with subsequent risk of PTDM. When occurring pretransplantation, the metabolic syndrome (27) and its components such as pretransplant hypertriglyceridemia and BMI (28), as well as prediabetes (17) have predicted increased risk for PTDM. Specifically pretransplantation insulin resistance, putatively the underlying pathophysiology of metabolic syndrome, was found to be a risk factor for PTDM (7).

Pancreatic beta cell dysfunction (reflected by high fasting proinsulin concentrations) has been shown to be a risk factor for PTDM (29) and these data are supported by genetic polymorphism studies. Kim et al (30) identified an association between single nucleotide polymorphisms within 10 genes of interleukins or their receptors as predictors of PTDM. Similarly, Tavira et al (31) demonstrated an association between KNNJ11 polymorphisms, hypothesized to impair insulin release from pancreatic beta cells, and PTDM.

Screening for posttransplant glucose abnormalities (Recommendation 3) is even more important in those patients identified to be at a higher risk of PTDM. In a prospective cohort of over 600 patients undergoing serial OGTTs, the vast majority of late PTDM were prediabetic at 3 months (32). Trials are needed to determine whether modifying established or novel risk factors can attenuate progression to PTDM.

Recommendation 5: Choose and Use Immunosuppression Regimens Shown to Have the Best Outcome for Patient and Graft Survival, Irrespective of PTDM Risk

Immunosuppression is the major modifiable risk factor for development of PTDM but risk versus benefit analysis is required to balance risk of developing PTDM versus rejection. Cole et al (33) demonstrated adverse graft survival after development of either rejection or PTDM, with development of both resulting in the worst outcomes. Therefore, no specific recommendation is made to advocate a definitive immunosuppressant strategy for allograft recipients based upon PTDM risk alone. Based on the current lack of evidence we also recommend caution in immunosuppressant adjustments in the event that PTDM develops, with a need to account for patient-specific risk factors such as immunological risk.

The DIRECT study confirmed the increased diabetogenicity of tacrolimus compared to cyclosporine postkidney transplantation in a randomized controlled trial, with no difference in adverse events (34). However, this was a 6-month trial and glycemic benefits need to be weighed against risk for long-term graft attrition. In addition, target tacrolimus levels were higher than the contemporary approach of reduced calcineurin inhibitor (CNI) exposure. A recent meta-analysis of 56 randomized controlled trials demonstrated less PTDM and better overall graft survival with CNI-minimization or avoidance strategies using new agents such as belatacept or tofacitinib (35). Results are awaited from groups evaluating alternative strategies such as selecting CNI (tacrolimus vs. cyclosporine) based on pretransplant PTDM risk (clinicaltrials.org: NCT01002339). There is limited evidence supporting conversion from tacrolimus to cyclosporine in established PTDM, both from previous literature (36) and a preliminary report of randomized trial data (37), but the benefits must be weighed against any risks associated with conversion. Late changes in immunosuppressive regimens may reverse PTDM without jeopardizing graft outcomes, but this requires further evaluation to ensure glycemic benefits outweigh allograft risks.

Johnston et al, analyzing data from the United States Renal Data System, found sirolimus were independently associated with increased risk for PTDM (38). Fewer reports have conflictingly found glycemic benefits after conversion from CNIs to sirolimus (39). There is no evidence to suggest any glycemic effects of anti-proliferative agents such as mycophenolate mofetil or azathioprine.

Steroid minimization is a common strategy to attenuate risk of PTDM. However, a beneficial effect of corticosteroid sparing strategies has not been demonstrated (40). A recent meta-analysis of corticosteroid withdrawal between 3 and 6 months after transplantation found no meaningful effect on PTDM incidence (41). Early corticosteroid withdrawal after a few days has shown decreased PTDM incidence, but this was only significant when the CNI used was cyclosporine compared to tacrolimus (42). Moreover, a mild increase in the incidence of acute rejection with corticosteroid sparing strategies might counterbalance the metabolic beneficial effect (41). The degree of glycemic burden from low-dose corticosteroid maintenance therapy is unclear and therefore steroid avoidance/withdrawal strategies require careful risk/benefit assessment in the context of long-term outcomes. The impact of steroid avoidance/withdrawal is all the more uncertain given the current use of lower CNI target levels and rapid weaning of corticosteroids. Split corticosteroid dosing may also reduce glycemic variability and peak hyperglycemia (43).

Data in relation to the impact of induction therapy are limited and no firm conclusions can be drawn. In a recent meta-analysis of five studies ($n = 492$ patients), the mAb alemtuzumab was found to be associated with a borderline lower risk of developing PTDM than IL-2 receptor antagonists (44). This could be due to CNI- and steroid-sparing strategies employed with alemtuzumab use or a diabetogenic effect of IL-2 receptor antagonists. Supporting the latter is a single-center retrospective study of 264 renal transplant recipients, where induction with basiliximab was associated with a significantly greater risk of developing PTDM compared to no induction (51.5% vs. 36.9%, $p = 0.017$) at 10 weeks posttransplantation (45).

Recommendation 6: Use Strategies for Prevention and Treatment Beyond Modification of Immunosuppressive Regimens

Prevention is ideal and guidance should be given to all potential transplant recipients regarding their risk of developing PTDM. Intervention when necessary can be in the form of nonpharmacological and/or pharmacological therapy. Sharif et al (46) demonstrated the potential for benefit from lifestyle modification in kidney allograft recipients with impaired glucose tolerance (13/25 patients reverted to normal glucose tolerance after median of 9 months, with only 1 progressing to PTDM). Thus, as observed in the general population (47), exercise and lifestyle modification may reduce the risk of patients with prediabetes developing PTDM. However, there remains a need for well-powered clinical trials to evaluate the

feasibility and efficacy of these interventions to prevent PTDM in a larger renal transplant population. Werzowa et al (48) reported a randomized controlled trial comparing safety and efficacy of vildagliptin (dipeptidylpeptidase-4 inhibitor) with pioglitazone (thiazolidinedione) or placebo in kidney allograft recipients with impaired glucose tolerance. Adverse events were equivalent in all three arms and both pioglitazone and vildagliptin produced comparable reduction in 2-h postprandial glucose levels. Metformin may be an attractive anti-hyperglycemic agent to reduce the likelihood of PTDM in high-risk individuals (49) but the benefits of metformin need to be weighed against the risks associated with metformin in the context of impaired renal function (e.g. lactic acidosis). However, this association has been the subject of critical analysis (50) and well-designed clinical trials are necessary to shed light on the benefit versus risk ratio in relation to metformin.

Lifestyle modification > oral anti-diabetic therapy > insulin is an appropriate stepwise approach for management of late-PTDM, but with immediate posttransplant hyperglycemia we recommend the *reverse* as the most appropriate management. Insulin is the only safe and effective agent in the context of high glucocorticoid doses and acute illness early posttransplant, but early and aggressive use of insulin may also have long-term benefits. In a randomized controlled trial, Hecking et al (12) demonstrated the benefit of early basal insulin therapy following detection of early posttransplant hyperglycemia (<3 weeks) at reducing subsequent odds of developing PTDM within the first year posttransplantation by 73%. A larger randomized controlled clinical trial (ITP-NODAT, clinicaltrials.org: NCT01683331) is currently evaluating whether these findings are reproducible in five centers recruiting over 300 patients. In addition, this study will determine whether early insulin therapy is feasible in patients who are hospitalized for a much shorter period than utilized in the original study. Treatment of posttransplantation hyperglycemia is in line with postoperative glucose management and, although representing a major shift from previous practice, consensus opinion was that this approach should be recommended but a glucose threshold for starting insulin was not specified (Figure 1). Although a relatively high glucose threshold of 200 mg/dL (evening or fasting) has been previously suggested, it may be reasonable to lower this threshold but further research is warranted before firm guidance can be issued.

The armamentarium of anti-diabetic therapy is increasing and individual pharmacological risk/benefit profiles must be evaluated in the context of transplantation (5,6,20). Dose adjustments or cessation of oral anti-diabetic agents in the context of renal allograft dysfunction should be individualized. Further work to understand the pathophysiology underlying PTDM development and progression should assist choice of pharmacological agents and form the basis

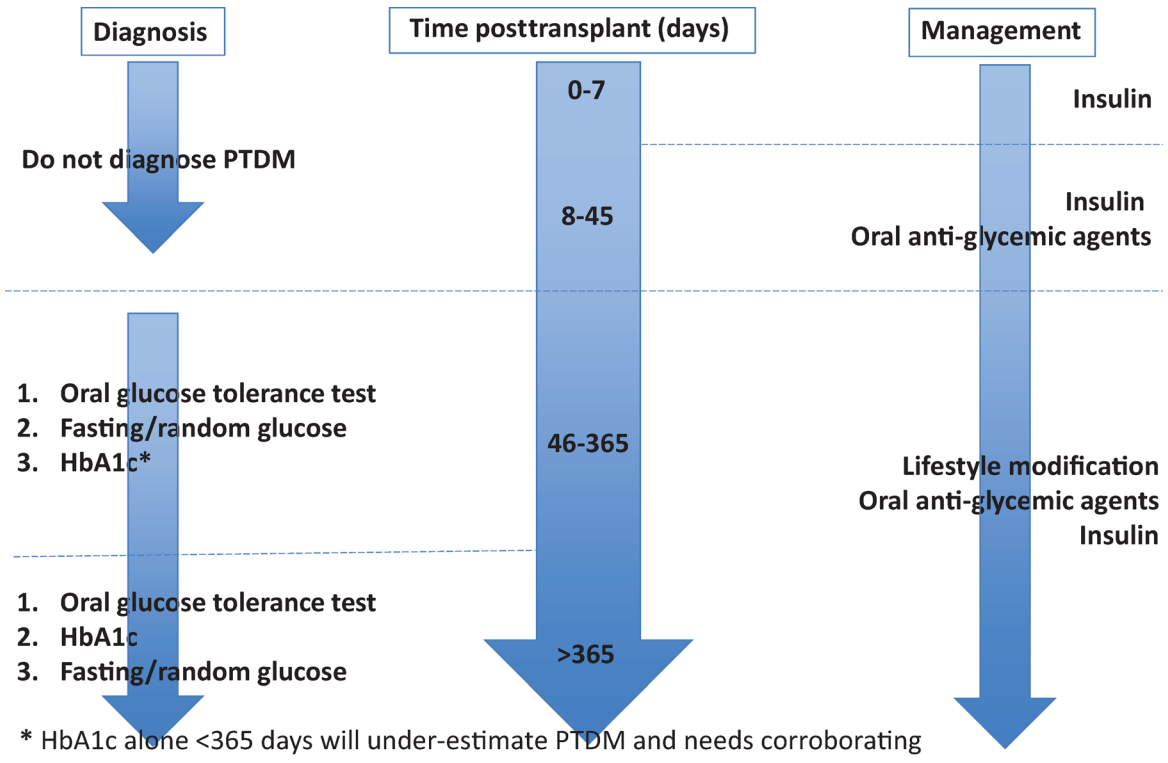

Figure 1: Flowchart highlighting updated diagnostic and management framework for posttransplantation diabetes mellitus.

of targeted clinical trials. Haidinger et al (51) have recently reported the first randomized controlled trial comparing vildagliptin with placebo for treatment of PTDM, demonstrating profound improvement in both 2-h postprandial glucose and HbA1c levels within 3 months. Halden et al (52) have also reported the short-term efficacy and safety of a different dipeptidylpeptidase-4 inhibitor (sitagliptin) in their randomized crossover study of 19 kidney allograft recipients with PTDM. Further clinical trials are warranted to attain a clinical evidence-base for the optimum agent or agent combinations for both safety and efficacy. For example, there are no data regarding the safety and/or efficacy of glucagon-like peptide 1 receptor agonists in the context of kidney transplantation.

The consensus group agreed there were inadequate data to recommend a hierarchy of anti-glycemic agents in this setting (53). Previous reluctance to use metformin posttransplantation was discussed in line with recent arguments focusing on its advantages (49). A research

Table 2: Currently active PTDM-related research studies registered on clinicaltrials.org

Clinicaltrials.org study identifier	Study completion	Title of research project	Study narrative
NCT01680185	August 2017	Sensor-Augmented Insulin-Pump Therapy in New-onset Diabetes After Transplantation (SAPT-NODAT)	Open label RCT testing benefits of intensive subcutaneous insulin via an insulin pump in comparison to standard of care (basal insulin)
NCT01683331	August 2017	A Clinical Trial to Prevent New Onset Diabetes After Transplantation (ITP-NODAT)	Open label RCT testing benefits of early insulin therapy for posttransplantation hyperglycemia in first week post-op
NCT01875224	August 2016	Comparison of NODAT in Kidney Transplant Patients Receiving Belatacept Versus Standard Immunosuppression	Open label RCT comparing glycemic benefits of *de novo* belatacept versus standard tacrolimus-based therapy
NCT01002339	June 2013	Optimum Immunosuppression in Renal Transplant Recipients. New Onset Diabetes After Transplantation (01-DMPT)	Open label RCT analyzing benefits and risks with 3 different *de novo* regimens in glycemic high-risk patients: (1) tacrolimus with rapid steroid withdrawal, (2) tacrolimus with steroid minimization, (3) cyclosporine with steroid minimization
NCT01928199	June 2015	Efficacy Study of Sitagliptin to Prevent New-Onset Diabetes After Kidney Transplant	Double blind RCT testing benefits of adding sitagliptin to preventing PTDM in recipient with transient posttransplantation hyperglycemia in first 72 h post-op
NCT01265537	December 2015	A Pilot Study Comparing the Use of Low-Target Versus Conventional Target Advagraf (Astellas)	Open label RCT comparing glycemic benefits of standard tacrolimus-based therapy with low-target tacrolimus regimen (+ thymoglobulin induction + early steroid withdrawal)
NCT01856257	October 2015	Safety and Efficacy of a Steroid-Free, Calcineurin Inhibitor-Free, Belatacept-Based Immunosuppressive Regimen	Open label RCT comparing glycemic benefits of a belatacept-based regimen (*de novo* or conversion) against tacrolimus-based therapy
NCT01431430	June 2015	VITamin D Supplementation in RenAL Transplant Recipients—VITALE	Double blind RCT to study the effect of low-dose versus high-dose colecalciferol supplementation on composite end point of PTDM, *de novo* cancer, cardiovascular disease and mortality
NCT01648218	June 2013	Insulin Therapy for Posttransplant Glucocorticoid Induced Hyperglycemia (PTHG)	Open label RCT to study which insulin therapy (NPH, Aspart or Glargine) is most effective to posttransplant hyperglycemia over a 48-h period during hospitalization for transplantation (PTDM or type 2 diabetes mellitus)
NCT01291030	May 2013	The Impact of Magnesium Supplementation on Insulin Resistance and Secretion in Renal Transplant Recipients	Open label RCT to assess effect of magnesium supplementation posttransplantation on insulin resistance and secretion indices (OGTT)
NCT01560572	April 2015	Steroid Free Immunosuppression or Calcineurin Inhibitor Minimization After Basiliximab Induction Therapy in Kidney Transplantation: Comparison With a Standard Quadruple Immunosuppression Regimen (Allegro)	Open label RCT investigating effects of steroid-free immunosuppression versus tacrolimus minimization at 6 months for secondary outcome of PTDM at 2 years (primary outcome is acute rejection and graft function)

RCT, randomized controlled trial; PTDM, posttransplantation diabetes mellitus; NPH, neutral protamine Hagedorn; OGTT, oral glucose tolerance test.

consortium of interested parties proposed a feasibility study looking at early use of metformin therapy for kidney allograft recipients. Clinical trials are warranted to assess safety and efficacy before metformin can be recommended as the anti-glycemic agent of choice.

There was widespread agreement that PTDM-related risks should be addressed in conjunction with other cardio-metabolic risk factors to reduce cardiovascular disease posttransplantation as comprehensively reviewed elsewhere (54).

Recommendation 7: Expand Basic, Translational and Clinical Research in the Field of PTDM to Resolve Unanswered Questions

Although our understanding of PTDM has been significantly enhanced over the last decade, continued research is essential to develop our clinical evidence base. The consensus group recommended greater emphasis on basic, translational and clinical research to resolve unanswered questions. Table 2 highlights studies currently in progress (as registered on clinicaltrials.org) in the field of PTDM and further targeted studies should be encouraged. From a practical standpoint, there is a need to collaborate and combine data linking fasting/postprandial glucose and HbA1c with end points including patient/graft survival, rates of malignancy, cardiovascular events and microvascular complications (such as retinopathy). Long-term complications relating to different glycemic indicators (e.g. HbA1c vs. 2-h glucose) are warranted. Shedding light on molecular mechanisms by which immunosuppressants affect beta cell function and insulin resistance could provide deeper insight into the pathophysiology and progression of PTDM, thus preexisting experimental models should be enhanced (55) and ''omics'' technology explored. From a clinical standpoint, trials to improve prediction of PTDM and delay or prevent PTDM are needed, including most appropriate risk factors to screen as part of routine clinical practice. We also need to determine whether improving glycemic control, especially early insulin treatment, improves long-term outcomes. Toward the latter aim we hope to facilitate clinical trials of PTDM prevention and management (pharmacological and/or nonpharmacological therapies), which are adequately powered to relevant end points such as cardiovascular events and mortality.

Conclusions

This Meeting Report summarizes opinion-based recommendations from the recently convened International Expert Panel review of PTDM and constitutes an overdue update to previous consensus guidance. Our recommendations, GRADE defined based upon expert opinion, reflect the perspective and knowledge acquired over the last decade and represents consensus from clinicians and researchers with active interest and expertise in the field. Our recommendations represent significant changes in practice and include: terminology revision from NODAT to PTDM, exclusion of transient posttransplant hyperglycemia from PTDM diagnosis, expansion of screening strategies (incorporating postprandial glucose and HbA1c) and opinion-based guidance regarding pharmacological therapy in light of recent clinical evidence.

Given the overall improvement in other areas of transplantation, PTDM now constitutes one of the most important complications associated with transplantation, associated with significant morbidity and mortality. The consensus group plan to submit updated guidelines encompassing and elaborating upon our opinion-based recommendations and intends to revise these recommendations in 3–5 years, with the anticipated benefit of new data and research.

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Disclosure

The authors of this manuscript have no conflicts of interest to disclose as described by the *American Journal of Transplantation*.

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