Association of Pretransplant Glycemic Control With Posttransplant Outcomes in Diabetic, Kidney-Transplant Recipients

MIKLOS Z. MOLNAR, MD, PHD1,2
EDMUND HUANG, MD3
JUNICHI HOSHINO, MD4
MAHESH KRISHNAN, MD, MPH, MBA5

ALLEN R. NISSENSON, MD5
CSABA P. KOVESDY, MD6,7
KAMYAR KALANTAR-ZADEH, MD, MPH, PHD1,3,4

OBJECTIVE—Observational studies have yielded inconsistent findings regarding the association of hemoglobin A1c (HbA1c) with survival in diabetic patients on dialysis. The association between pretransplant glycemic control and short- and long-term posttransplant outcomes in kidney-transplant recipients is not clear.

RESEARCH DESIGN AND METHODS—Linking the 5-year patient data of a large dialysis organization (DaVita) to the Scientific Registry of Transplant Recipients, we identified 2,872 diabetic dialysis patients who underwent first kidney transplantation. Mortality or graft failure and delayed graft function (DGF) risks were estimated by Cox regression (hazard ratio [HR]) and logistic regression (odds ratio), respectively.

RESULTS—Patients were 53 ± 11 years old and included 36% women and 24% African Americans. In our fully adjusted model, allograft failure–censored, all-cause death HR and 95% CI for time-averaged pretransplant HbA1c categories of 7 to <8%, 8 to <9%, 9 to 10%, and ≥10%, compared with 6 to <7% (reference), were 0.89 (0.59–1.36), 2.06 (1.31–3.24), 1.41 (0.73–2.74), and 3.43 (1.56–7.56), respectively; and graft failure–censored cardiovascular death HR was 0.38 (0.13–1.05), 1.78 (0.69–4.55), 1.59 (0.44–5.76), and 4.28 (0.85–21.64), respectively. We did not find any difference in risk of death–censored graft failure or DGF with different pretransplant HbA1c levels.

CONCLUSIONS—Poor pretransplant glycemic control appears associated with decreased posttransplant survival in kidney-transplant recipients, whereas allograft outcomes may not be affected.

Diabetes is a potent cardiovascular risk factor in the general population as well as in those undergoing maintenance dialysis and kidney-transplant recipients (1,2). Clinical trials have shown that tight glycemic control decreases the risk of developing retinopathy, nephropathy, and neuropathy in the general population (3,4). Furthermore, glycemic control, as measured by glycylated hemoglobin (HbA1c), is a predictor of cardiovascular complications, including myocardial infarctions and hospitalizations for coronary artery disease (2). Expert groups have recommended that diabetic dialysis patients should follow the American Diabetes Association (ADA) guidelines; however, there is no consistent evidence to support these recommendations for patients with end-stage renal disease (5–7).

In concordance with the ADA guidelines, the Kidney Disease Outcomes Quality Initiative (K-DOQI) recommendations, last updated in 2007, state that “Target HbA1c for people with diabetes should be <7%, irrespective of presence or absence of CKD” (8).

Large observational studies with differing methodologies reached somewhat contrasting conclusions regarding the association of glycemic control with survival in diabetic maintenance hemodialysis and peritoneal dialysis patients. Recently, a large randomized trial has indicated that intensive glucose lowering in patients with type 2 diabetes did not reduce the risks of cardiovascular disease, the most common source of end-stage renal disease mortality (9). Additionally, Williams et al. (10) reported a higher risk for death only in type 2 diabetic hemodialysis patients with HbA1c levels >11% (11). Shurraw et al. (12) found higher casual glucose and HbA1c levels were not associated with mortality in maintenance hemodialysis patients with or without diabetes. In contrast, we reported that after adjusting for potential confounders, higher HbA1c values were incrementally associated with higher death risks in patients on maintenance dialysis (13).

Furthermore, in peritoneal dialysis patients, only poor glycemic control (HbA1c ≥8% and/or glucose ≥300 mg/dL) was incrementally associated with lower survival (14). Alas, mortality is only one measure of the deleterious impact of poor glycemic control. Other potential benefits of glycemic control include slowing the rate of progression of micro- and macrovascular disease, decreasing the presence of nonfatal strokes and myocardial infarctions, and slowing the rate progression of nephropathy. These factors have a strong impact on survival in kidney-transplant recipients.

To the best of our knowledge, no study has examined the association between pretransplant glycemic control and either short-term outcome, such as delayed graft function (DGF), or long-term outcomes, such as mortality and graft failure after kidney transplantation. We...
hypothesized that higher pretransplant HbA1c, during the dialysis period prior to kidney transplantation is associated with worse posttransplant patient and graft survival and with DGF in a large prospective cohort of incident kidney-transplant recipients across the United States.

RESEARCH DESIGN AND METHODS

Patients
We linked data on all kidney-transplant recipients listed in the Scientific Registry of Transplant Recipients (SRTR) up to June 2007 to a list of individuals with CKD who underwent maintenance hemodialysis or peritoneal dialysis treatment from July 2001 to June 2006 in one of the outpatient dialysis facilities of a U.S.-based large dialysis organization (DaVita Inc., prior to its acquisition of former Gambro dialysis facilities) using patients’ social security numbers. The study was approved by the Institutional Review Boards of both Los Angeles Biomedical Research Institute at Harbor-UCLA and DaVita Clinical Research. Inclusion criteria were patients who had been undergoing dialysis for at least 90 days, had a history of diabetes, and had at least one HbA1c measurement in the first quarter of entry into the cohort.

Clinical and demographic measures
The creation of the national DaVita dialysis patient cohort has been described previously (14,15). To minimize measurement variability, all repeated measures for each patient during any given calendar quarter, i.e., over a 13-week interval, were averaged, and the summary estimate was used in all models. Average values were obtained from up to 20 calendar quarters (q1–q20) for each laboratory and clinical measure for each patient for up to 6 years of follow-up. The first (baseline) studied quarter for each patient was the calendar quarter in which the patient’s dialysis vintage was >90 days. Demographic and laboratory data were collected, with information on age, sex, race, type of insurance, marital status, height, posthemodialysis dry weight (to calculate averaged BMI), and dialysis vintage. Dialysis vintage was defined as the duration of time between the first day of dialysis treatment and the day of kidney transplantation.

Laboratory measures
All HbA1c values were measured using the Roche Cobas Integra 800 whole-blood immune-turbidimetric assay (standardized according to the Diabetes Complications Control Trial/National Glycohemoglobin Standardization Program) performed by a single laboratory (DaVita Laboratory, Deland, FL). Most laboratory values were measured monthly, including serum urea, creatinine, albumin, calcium, phosphorus, bicarbonate, and total iron binding capacity. Serum ferritin and intact parathyroid hormone were measured at least quarterly. Hemoglobin was measured at least monthly in essentially all patients and weekly to biweekly in most patients. Most blood samples were collected predialysis, with the exception of postdialysis serum urea nitrogen to calculate urea kinetics. HbA1c was usually measured quarterly or semiannually. We divided patients into seven a priori–defined categories based on HbA1c values: <5%, ≥10%, and 1% increments in between, to examine the dose-response association between HbA1c categories and outcome risk.

Statistical methods
Data were summarized using proportions, means (±SD) or medians, and interquartile ranges as appropriate. We examined P values for trends across HbA1c categories. For all-cause and cardiovascular mortality and graft failure, defined as reinitiation of dialysis treatment or retransplantation, time to event was used in all survival analyses. For DGF, defined as the need for any dialysis therapy in the first week after transplantation, time to event was not accounted for. Survival analyses to calculate hazard ratio (HR) and 95% CI of death or graft failure used Cox proportional hazards regression. In the mortality analyses, the patients were followed until event (death) or censoring (graft failure or end of follow-up period), whichever happened first. Our uncensored, all-cause mortality analysis patients were followed until event (death) or censoring (graft failure or end of follow-up period), whichever happened first. In the graft failure analyses, the patients were followed until event (graft failure) or censoring (death or end of follow-up period), whichever happened first. In the combined outcome analyses, patients were followed until event (death or graft failure) or censoring (end of follow-up period), whichever happened first. Logistic regression models were used to estimate the odds ratio and 95% CI of posttransplant DGF.

For each regression analysis, four levels of multivariate adjustment were examined: I) an unadjusted model that included HbA1c categories (reference, HbA1c 6 to <7%) as the predictor; II) case mix–adjusted models that included the above plus age, sex, recipient race/ethnicity (African Americans and other self-categorized blacks, non-Hispanic whites, Asians, Hispanics, and others), dialysis vintage (<6 months, 6 months to 2 years, 2 to <5 years, and ≥5 years), primary insurance (Medicare, Medicaid, private, and others), marital status (married, single, divorced, widowed, and other or unknown), standardized mortality ratio of the dialysis clinic during entry quarter, dialysis dose as indicated by Kt/V (single pool), presence or absence of a hemodialysis catheter, residual renal function during the entry quarter and eight co-morbidities (atherosclerotic heart disease, congestive heart failure, cancer, chronic obstructive pulmonary disease, cerebrovascular disease, hypertension, peripheral vascular disease, and tobacco use); III) malnutrition inflammation complex syndrome (MICS)–adjusted models, which included all of the above covariates plus 11 surrogates of nutritional status and inflammation measured during the last calendar quarter before transplantation, including BMI and 10 laboratory variables, i.e., normalized protein catabolic rate as an indicator of daily protein intake, also known as the normalized protein nitrogen appearance (nPNA), and serum or blood concentrations of total iron binding capacity), ferritin, phosphorus, calcium, bicarbonate, peripheral white blood cell count (WBC), lymphocyte percentage, albumin and hemoglobin; and IV) case mix–, MICS–, and transplant data–adjusted models included all of the above plus seven transplant–related variables: (1) donor type (deceased or living), (2) donor age, (3) donor sex, (4) panel-reactive antibody titer (last value prior to transplant), (5) number of HLA mismatches, (6) cold ischemia time, and (7) DGF (except when DGF was a dependent variable in our logistic regression models). Nonlinear association between pretransplant, time-averaged HbA1c and posttransplant outcomes was assessed using fractional polynomials and restricted cubic splines. Based on goodness of fit of these models, we used a model with two degrees of freedom (knot in all models was 6.59). All analyses were carried out with SAS version 9.1 (SAS Institute, Inc., Cary, NC) and STATA version 11.1 (STATA Corporation, College Station, TX).

RESULTS—The original 5-year (July 2001–June 2006) national database of
all DaVita patients included 164,789 adult subjects. Out of 65,386 DaVita patients who were identified in the SRTR database, 17,629 had undergone one or more kidney transplants during their lifetime, of which 14,508 dialysis patients had undergone kidney transplantation for the first time. From these 14,508 dialyzed patients, we excluded patients without diabetes (n = 9,482) and patients who did not have HbA1c measured (n = 2,154). We examined the remaining 2,872 patients who underwent first kidney transplantation during the observation period and who were followed until death, graft failure, loss of follow-up, or survival until 30 June 2007 (Supplementary Fig. 1). There were 331 deaths (11.5%) and 191 graft failures (6.7%) irrespective of subsequent deaths. The median follow-up time was 736 days (interquartile range, 353–1,216 days).

Table 1 shows the clinical, demographic and laboratory data of the 2,872 transplanted patients across seven categories of HbA1c. Patients with higher HbA1c were more likely to be women and to be younger, and to have lower dialysis duration, lower serum creatinine level, and higher WBC level. The crude rates of mortality, graft failure, and DGF were not different between the groups (Table 2).

Supplementary Table 1 shows the calculated HRs of all-cause and cardiovascular death and/or graft failure for different HbA1c categories. Case mix-, MICS-, and transplant data-adjusted graft failure censored all-cause death HRs and 95% CIs for time-averaged pretransplant HbA1c categories of 7 to <8%, 8 to <9%, 9 to 10%, and ≥10%, compared with 6 to <7% (reference), were 0.89 (0.59–1.36), 2.06 (1.31–3.24), 1.41 (0.73–2.74), and 3.43 (1.56–7.56), respectively. Fig. 1A shows cubic spline models for the association of the entire range of pretransplant HbA1c with posttransplant cardiovascular mortality with posttransplant cardiovascular mortality that were consistent with the findings in Supplementary Table 1. Fig. 1B shows cubic spline models for the association of the entire range of pretransplant HbA1c with posttransplant cardiovascular mortality that were consistent with the findings in Supplementary Table 1. Fig. 1C shows cubic spline models for the association of the entire range of pretransplant HbA1c with posttransplant cardiovascular mortality that were consistent with the findings in Supplementary Table 1. We did not find any difference in the risk of all-cause death–censored graft failure with different pretransplant HbA1c levels.

To examine the association of pretransplant HbA1c levels with posttransplant DGF, multivariate logistic regression analyses were performed using the same covariates as in the Cox models. Compared with HbA1c levels between 6 and <7%, patients with HbA1c levels <5%, 5 to <6%, 6 to <8%, 8 to <9%, 9 to 10%, and ≥10% had similar risks of DGF (Supplementary Table 1).

**CONCLUSIONS**—In 2,872 kidney-transplant recipients with comprehensive pretransplant data during hemodialysis treatment who were followed for up to 6 years posttransplantation, poor glycemic control appears to be associated with higher all-cause and cardiovascular mortality. Pretransplant HbA1c level was not a predictor of either posttransplant graft failure or DGF. These findings may have important clinical implications, especially since they imply mild hyperglycemia during the dialysis period may not be a risk factor for negative posttransplant short-term and long-term outcomes.

It is currently not known what the ideal level of glycemic control is in dialysis patients (16,17). The literature on the relationship between glycemic control and survival in CKD populations is somewhat limited. In a cohort of 840 nondiabetic patients with moderate CKD who participated in the Modification of Diet in Renal Disease trial, HbA1c was a predictor of all-cause mortality (18). However, a recent study was unable to demonstrate any association between HbA1c and 1-year survival (7). This contrasts with several other observational studies. Wu et al. (19) studied 137 hemodialysis patients with type 2 diabetes and reported that the cumulative survival was lower in the group with poor glycemic control (13). In kidney-transplant recipients, pretransplant diabetes, maximal glucose levels, and insulin treatment were independently associated with higher rates of mortality (1). However, until now, it has been unclear if poor glycemic control during the dialysis period affected posttransplant outcomes.

Based on our analyses, pretransplant, time-averaged HbA1c levels ≥8% were associated with increased all-cause and cardiovascular mortality. There are several possible mechanisms that might explain the relationship between poor glycemic control during the dialysis period and worse posttransplant survival of kidney-transplant recipients. Diabetes after transplantation is a predictor of mortality in some (20), but not all, studies (1). It is also associated with different risk factors of mortality, such as higher infection rates or other diabetes complications (21). Poor glycemic control might induce macrovascular complications, possibly secondary to the generation of advanced glycation end products, and hence shorten the survival of these patients. Moreover, in renal transplant recipients, high HbA1c is associated with chronic inflammation (22), which is a known predictor of mortality, graft failure (23).

In our contemporary study, the pretransplant HbA1c level was not a predictor of posttransplant graft failure or DGF. Similar results were found in studies conducted in kidney-transplant recipients that examined new-onset diabetes after transplantation, which was not a predictor of graft loss in some studies (24). A potential explanation for the lack of association between poor pretransplant and posttransplant glycemic control and increased risk of allograft loss is the relatively short follow-up time. Poor glycemic control is associated with micro- and macrovascular complication, but the development of these complications in the allografts may take ≥6 years. It is possible that studies with longer follow-up time may find significant associations between poor glycemic control and graft loss.

Our study should be qualified for several potential limitations. Like all observational studies, ours too cannot prove causality. Repeated posttransplant measures of HbA1c or other laboratory variables and immunosuppressive and other medical regimens were not available in the SRTR database, but in the full model, we did adjust for a number of transplant-related variables. A potential limitation of
## Table 1—Baseline characteristics of 2,872 dialysis patients who underwent renal transplantation between July 2001 and June 2006

<table>
<thead>
<tr>
<th>HbA1c (%)</th>
<th>&lt;5</th>
<th>5 to &lt;6</th>
<th>6 to &lt;7</th>
<th>7 to &lt;8</th>
<th>8 to &lt;9</th>
<th>9 to &lt;10</th>
<th>≥10</th>
<th>P for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (%)</td>
<td>171 (6)</td>
<td>708 (25)</td>
<td>873 (30)</td>
<td>591 (20)</td>
<td>315 (11)</td>
<td>154 (5)</td>
<td>60 (2)</td>
<td>NA</td>
</tr>
<tr>
<td>Age (years) (mean ± SD)</td>
<td>52 ± 13</td>
<td>55 ± 10</td>
<td>55 ± 10</td>
<td>54 ± 10</td>
<td>50 ± 11</td>
<td>48 ± 12</td>
<td>46 ± 11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Gender (% women)</td>
<td>41</td>
<td>33</td>
<td>35</td>
<td>34</td>
<td>40</td>
<td>45</td>
<td>45</td>
<td>0.01</td>
</tr>
<tr>
<td>Race (% African American)</td>
<td>24</td>
<td>26</td>
<td>23</td>
<td>23</td>
<td>18</td>
<td>27</td>
<td>35</td>
<td>0.07</td>
</tr>
<tr>
<td>BMI (kg/m²) (mean ± SD)</td>
<td>27.3 ± 5.3</td>
<td>28.3 ± 5.0</td>
<td>28.5 ± 5.6</td>
<td>28.2 ± 5.5</td>
<td>27.0 ± 5.6</td>
<td>27.6 ± 5.4</td>
<td>26.8 ± 5.7</td>
<td>0.004</td>
</tr>
<tr>
<td>Presence of ischemic heart disease (%)</td>
<td>7</td>
<td>16</td>
<td>17</td>
<td>16</td>
<td>14</td>
<td>14</td>
<td>15</td>
<td>0.06</td>
</tr>
<tr>
<td>Presence of congestive heart failure (%)</td>
<td>12</td>
<td>15</td>
<td>17</td>
<td>21</td>
<td>18</td>
<td>18</td>
<td>15</td>
<td>0.09</td>
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<td>Presence of hypertension (%)</td>
<td>78</td>
<td>84</td>
<td>80</td>
<td>81</td>
<td>81</td>
<td>82</td>
<td>77</td>
<td>0.49</td>
</tr>
<tr>
<td>Presence of cerebrovascular events (%)</td>
<td>1</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>10</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>Presence of peripheral vascular disease (%)</td>
<td>5</td>
<td>7</td>
<td>9</td>
<td>8</td>
<td>7</td>
<td>8</td>
<td>10</td>
<td>0.78</td>
</tr>
<tr>
<td>Presence of chronic obstructive pulmonary disease (%)</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0.76</td>
</tr>
<tr>
<td>Presence of cancer (%)</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0.03</td>
</tr>
<tr>
<td>Tobacco use (%)</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>0.24</td>
</tr>
<tr>
<td>Dialysis vintage (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.002</td>
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<tr>
<td>0–6 months</td>
<td>11</td>
<td>8</td>
<td>9</td>
<td>8</td>
<td>9</td>
<td>13</td>
<td>18</td>
<td>—</td>
</tr>
<tr>
<td>6–24 months</td>
<td>24</td>
<td>30</td>
<td>30</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>31</td>
<td>40</td>
</tr>
<tr>
<td>2–5 years</td>
<td>37</td>
<td>42</td>
<td>45</td>
<td>43</td>
<td>43</td>
<td>36</td>
<td>25</td>
<td>—</td>
</tr>
<tr>
<td>≥5 years</td>
<td>27</td>
<td>20</td>
<td>16</td>
<td>17</td>
<td>16</td>
<td>20</td>
<td>17</td>
<td>—</td>
</tr>
<tr>
<td>nPCR (g/kg/day) (mean ± SD)</td>
<td>1.01 ± 0.27</td>
<td>1.07 ± 0.27</td>
<td>1.06 ± 0.25</td>
<td>1.06 ± 0.26</td>
<td>1.04 ± 0.25</td>
<td>1.00 ± 0.22</td>
<td>0.99 ± 0.24</td>
<td>0.06</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL) (mean ± SD)</td>
<td>9.8 ± 2.9</td>
<td>9.8 ± 3.0</td>
<td>9.2 ± 2.9</td>
<td>9.1 ± 2.8</td>
<td>8.8 ± 2.6</td>
<td>8.9 ± 2.8</td>
<td>8.3 ± 2.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Blood hemoglobin (g/dL) (mean ± SD)</td>
<td>12.1 ± 1.4</td>
<td>12.3 ± 1.2</td>
<td>12.3 ± 1.2</td>
<td>12.3 ± 1.2</td>
<td>12.3 ± 1.1</td>
<td>12.3 ± 1.2</td>
<td>12.5 ± 1.1</td>
<td>0.13</td>
</tr>
<tr>
<td>WBC (×10³/L) (mean ± SD)</td>
<td>6.4 ± 1.9</td>
<td>6.9 ± 2.1</td>
<td>7.1 ± 2.1</td>
<td>7.3 ± 2.1</td>
<td>7.4 ± 2.2</td>
<td>7.3 ± 1.9</td>
<td>7.3 ± 1.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Number of HLA mismatch (median [IQR])</td>
<td>4 (3–5)</td>
<td>4 (2–5)</td>
<td>4 (3–5)</td>
<td>4 (2–5)</td>
<td>4 (2–5)</td>
<td>4 (2–5)</td>
<td>4 (3–5)</td>
<td>0.26</td>
</tr>
<tr>
<td>PRA (%)</td>
<td>7</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>4</td>
<td>5</td>
<td>4</td>
<td>0.39</td>
</tr>
<tr>
<td>PRA (% median [IQR])</td>
<td>0 (0–6)</td>
<td>0 (0–0)</td>
<td>0 (0–1)</td>
<td>0 (0–0)</td>
<td>0 (0–1)</td>
<td>0 (0–2)</td>
<td>0 (0–4)</td>
<td>0.63</td>
</tr>
<tr>
<td>Donor age (years) (mean ± SD)</td>
<td>39 ± 14</td>
<td>41 ± 16</td>
<td>41 ± 16</td>
<td>41 ± 15</td>
<td>39 ± 15</td>
<td>38 ± 15</td>
<td>37 ± 17</td>
<td>0.005</td>
</tr>
<tr>
<td>Donor gender (% women)</td>
<td>44</td>
<td>50</td>
<td>49</td>
<td>49</td>
<td>45</td>
<td>48</td>
<td>41</td>
<td>0.49</td>
</tr>
<tr>
<td>Donor type (% living)</td>
<td>34</td>
<td>28</td>
<td>31</td>
<td>30</td>
<td>32</td>
<td>34</td>
<td>45</td>
<td>0.15</td>
</tr>
<tr>
<td>Cold ischemia time (hours) (median [IQR])</td>
<td>14 (7–19)</td>
<td>14 (9–20)</td>
<td>14 (7–20)</td>
<td>14 (9–20)</td>
<td>14 (7–19)</td>
<td>14 (8–20)</td>
<td>14 (3–15)</td>
<td>0.40</td>
</tr>
</tbody>
</table>

Data are presented mean ± SD or median and interquartile range (IQR) as appropriate. nPCR, normalized protein catabolic rate; PRA, panel-reactive antibody.
our study is that HbA1c is not a good re-

flection of blood glucose levels in indi-

viduals with advanced chronic kidney
disease. It may be difficult to accurately

assess glycemic control in this population
because of changes in erythrocyte survival

in renal failure and the effects of erythro-

poiesis-stimulating agents on HbA1c lev-

els (25). We did not have access to data
pertaining to death after graft loss, which

is another important outcome. Patients

who did not have measured HbA1c were

excluded from the analyses. The excluded

patients may have been different from
those included in our study, which may

have biased our results. We have tested
this hypothesis and the only clinically sig-

nificant difference was in the number of

graft failures (Supplementary Table 2).
The proportion of graft failures was more

than twice higher in excluded patients than

the group included. It is possible that the

lower HbA1c levels in the included patients

were the reason why we did not detect any

association between high HbA1c level and
graft failure. To our knowledge this is the

first study examining the association be-
tween pretransplant HbA1c levels and post-

transplant short- and long-term outcomes.
Strengths of this study include the high

number of patients, the relatively long

follow-up time, and multilevel adjust-

ment, which includes several important

pretransplant measures.

In our large and contemporary na-
tional database of 2,872 kidney-transplant

Table 2—Outcomes of 2,872 dialysis patients who underwent renal transplantation between July 2001 and June 2006

<table>
<thead>
<tr>
<th>HbA1c (%)</th>
<th>&lt;5</th>
<th>5 to &lt;6</th>
<th>6 to &lt;7</th>
<th>7 to &lt;8</th>
<th>8 to &lt;9</th>
<th>9 to &lt;10</th>
<th>≥10</th>
<th>P for trend</th>
</tr>
</thead>
</table>

Values in brackets indicate the crude death and cardiovascular death rate, crude graft failure rate, and crude DGF rate in the indicated group during the 6 years of observation. CV, cardiovascular.

Figure 1—HR (95% CI) of posttransplant, graft failure–censored all-cause death (A), posttransplant, all-cause death (B), posttransplant, graft failure–censored cardiovascular death (C), and death-censored graft failure (D) across the entire range of the pretransplant, time-averaged HbA1c using fully adjusted Cox regression analyses in 2,872 long-term hemodialysis transplant patients who underwent renal transplantation and who were observed over a 6-year observation period (July 2001–June 2007).
Recipients, a pretransplant, time-averaged HbA1c ≥ 8% appears to be associated with higher all-cause and cardiovascular mortality. Pretransplant HbA1c levels were not predictive of posttransplant graft failure or DGF. Clinical trials are needed to better define optimal target HbA1c levels in dialysis patients.

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