Glycemic Control and Survival in Peritoneal Dialysis Patients with Diabetes Mellitus

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Summary

Background and objectives The optimal target for glycemic control has not been established for diabetic peritoneal dialysis (PD) patients.

Design, setting, participants, & measurements We examined mortality-predictability of hemoglobin A1c random serum glucose in a contemporary cohort of diabetic PD patients treated in DaVita dialysis clinics July 2001 through June 2006 with follow-up through June 2007.

Results We identified 2798 diabetic PD patients with A1c data. Serum glucose correlated with A1C (r = 0.51). Adjusted all-cause death hazard ratio and 95% confidence interval for baseline A1c increments of 7.0 to 7.9%, 8.0 to 8.9%, 9.0 to 9.9%, and ≥10%, compared with 6.0 to 6.9% (reference), were 1.13 (0.97 to 1.32), 1.05 (0.88 to 1.27), 1.06 (0.84 to 1.34), and 1.48 (1.18 to 1.86); and for time-averaged A1c values were 1.10 (0.96 to 1.27), 1.28 (1.07 to 1.53), 1.34 (1.05 to 1.70), and 1.81 (1.33 to 2.46), respectively. The A1c-mortality association was modified by hemoglobin level such that higher all-cause mortality was evident only in non-anemic patients. Similar but non-significant trends in cardiovascular death risk was found across A1c increments. Adjusted all-cause death HR for time-averaged blood glucose 150 to 199, 200 to 249, 250 to 299, and ≥300 mg/dl, compared with 60 to 99 mg/dl (reference), were 1.02 (0.70 to 1.47), 1.12 (0.77 to 1.63), 1.45 (0.97 to 2.18), and 2.10 (1.37 to 3.20), respectively.

Conclusions Poor glycemic control appears associated incrementally with higher mortality in PD patients. Moderate to severe hyperglycemia is associated with higher death risk especially in certain subgroups.

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Introduction

Diabetes mellitus (DM) is a potent cardiovascular risk factor in the general population as well as in those undergoing maintenance dialysis (1–5). Clinical trials have shown that tight glycemic control decreases the risk of developing retinopathy, nephropathy, and neuropathy in the general population (6,7). Furthermore, glycemic control—as measured by glycosylated hemoglobin (A1c)—is a predictor of cardiovascular complications, including myocardial infarctions and hospitalizations for coronary artery disease (1,8). Expert groups have recommended that diabetic dialysis patients should follow the American Diabetes Association guidelines; however, there is no consistent evidence to support these recommendations for patients with end-stage renal disease (ESRD), and the data for patients treated with peritoneal dialysis (PD) are even more limited (9–12).

There are several issues unique to the dialysis population that mandates a separate examination of the glycemic control on outcomes in this population. Chronic kidney disease is associated with insulin resistance and, in advanced kidney disease, decreased insulin degradation (13). Moreover, it has been difficult to accurately assess glycemic control in this population due to alterations in insulin metabolism and changes in red blood cell survival that lead to competing effects on measurements of glycemic control (13,14). Finally, tight glycemic control has potential problems for patients on dialysis, and there is some evidence that A1c may not be as predictive of glycemic control in dialysis patients (15).

Only a handful of studies have examined the association between A1c and clinical outcome in the dialysis population (11,16–20). Furthermore, only three of these studies included ≥150 subjects and none of these larger studies included patients treated with PD (11,20,21). The issue of glycemic control may be particularly important for PD patients for at least two reasons. First, several observational studies have shown a higher risk for death in older diabetic PD patients, compared with those treated with hemodialysis (22,23). Second, dialysis solutions that are used by PD patients contain glucose at concentrations ranging from 1360 to 3860 mg/dl; obligatory glucose absorption from these solutions contributes to hyper-

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glycemia in PD patients. Despite the apparent importance of glucose metabolism in PD patients, to our knowledge only one study has examined the association between A1c and clinical outcome in PD patients (24). Hence, we undertook this study to examine the predictive value of glycemic control on all-cause and cardiovascular mortality in a large, contemporary cohort of PD patients.

Materials and Methods

Patients

We extracted, refined, and examined data from all individuals with ESRD who underwent PD treatment July 2001 through June 2006 in any one of the 580 outpatient dialysis facilities of DaVita, a large dialysis organization in the United States (before its acquisition of units owned by Gambro). The study was approved by relevant Institutional Review Committees. Inclusion criteria were patients who had been undergoing dialysis for at least 90 days, were treated with PD at the time of entry into the cohort, had a history of diabetes mellitus, and had at least one A1c measurement in the first quarter of entry into the cohort. The original 5-year (July 2001 through June 2006) national database of all DaVita dialysis patients included 164,789 cumulative subjects; of these, 10,528 were treated with PD.

Clinical and Demographic Measures

The creation of the cohort has been described previously (25,26). To minimize measurement variability, all repeated measures for each patient during any given calendar quarter (q), that is, 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 through q20) for each laboratory and clinical measure for each patient over the 6-year cohort period. The first (baseline) studied quarter for each patient was the calendar quarter in which patient's vintage was >90 days. The presence or absence of diabetes at baseline was obtained from DaVita data. Histories of tobacco smoking and pre-existing comorbid conditions were obtained by linking the DaVita database to the Medical Evidence Form 2728 of the United States Renal Data System (USRDS) and categorized into 10 comorbid conditions: ischemic heart disease, congestive heart failure, history of cardiac arrest, history of myocardial infarction, pericarditis, cardiac dysrhythmia, cereberovascular events, peripheral vascular disease, chronic obstructive pulmonary disease, and cancer (27).

Patients were followed for outcomes through June 30, 2007. The recorded causes of death were obtained from the USRDS, and cardiovascular death was defined as death due to myocardial infarction, cardiac arrest, heart failure, cerebrovascular accident, and other cardiac causes.

Laboratory Measures

Blood samples were drawn using uniform techniques in all dialysis clinics and were transported to the DaVita Laboratory in Deland, Florida, within 24 hours. All laboratory values, including A1c, were measured by automated and standardized methods. Most laboratory values were measured monthly. A1c was usually measured quarterly or semiannually. The median number of A1c measurements per patient was 5 (interquartile range, 3 to 9). We divided patients into seven *a priori* categories based on A1c values <5%, ≥10%, and 1% increments in between, to examine the "dose-response" association between A1c categories and death risk. Additional analyses were performed after dividing the population into two groups of A1C ≥7% and <7%.

Epidemiologic Methods and Statistical Analyses

Intent-to-treat analyses were performed to mitigate the impact of selection bias due to transfer of PD patients to hemodialysis. Survival analyses with Kaplan-Meier log-rank test and Cox proportional hazard regression with repeated quarterly measures were used to examine whether glycemic control predicted survival for up to 6 years of follow-up. The primary analysis examined the association between baseline A1C and glucose with all-cause mortality, with cardiovascular mortality as a secondary outcome measure. We also performed exploratory analyses in subgroups of patients based on age, gender, race, dialysis vintage, serum albumin category (≤3.8 or >3.8 g/dl), and anemia (serum hemoglobin ≤11 or >11 g/dl and serum ferritin ≤500 or >500 ng/ml). We also analyzed the predictive value of time-averaged A1c. For each analysis, including subgroup analyses, three models were examined:

1. Unadjusted model that included mortality data, A1c categories, and entry calendar quarter (q1 through q20);
2. Case-mix adjusted model that included all of the above plus age, gender, race/ethnicity (African Americans and other self-categorized blacks, non-Hispanic Caucasians, Asians, Hispanics, and others), ten pre-existing comorbid conditions, history of tobacco smoking, categories of dialysis vintage (<6 months, 6 months to 2 years, 2 to 5 years, and ≥5 years), primary insurance (Medicare, Medicaid, private, and others), and marital status (married, single, divorced, widowed, and other or unknown);
3. Case-mix plus malnutrition-inflammation-complex syndrome (MICS) adjusted model which included all of the covariates in the case-mix model as well as 11 surrogates of nutritional status and inflammation, including body mass index, and 10 laboratory surrogates with known association with clinical outcomes in hemodialysis patients (26) including serum levels of albumin, total iron-binding capacity, ferritin, creatinine, phosphorus, calcium, bicarbonate, blood white blood cell count, lymphocyte percentage, and hemoglobin.

Missing covariate data were imputed by the mean or median of the existing values as appropriate. For all analysis, two-sided P values are reported and results considered statistically significant if $P < 0.05$. All statistical analyses were carried out with the SAS, version 9.1 (SAS Institute, Inc., Cary, NC).

Results

Over the 5-year period (July 2001 through June 2006), 164,789 adult subjects received dialysis treatment in units owned by DaVita Inc. (Supplemental Figure e1)—of these 10,528 patients were undergoing PD at the time of entry

Table 1. Demographic, clinical, and laboratory characteristics in studied group of 2798 with and 1291 diabetic PD patients without A1C measured in the calendar quarter of entry into the cohort

Variables	PD Patients with DM and A1C Measurement (n = 2798)	PD Patients with DM and No A1C Measurement (n = 1291)	P
Age (years)	58 ± 13	59 ± 13	0.20
Gender (% women)	44	46	0.17
Diabetes as cause ESRD (%)	85	75	<0.001
Race/ethnicity (%)			0.06
Caucasian	53	55	
African American	20	22	
Hispanic	16	13	
Vintage (time on dialysis, %)			<0.001
<6 months	71	60	
6 to 24 months	16	23	
2 to 5 years	11	13	
>5 years	2	4	
Primary insurance			<0.001
Medicare (%)	59	64	
Body mass index (kg/m^2)	27.0 ± 4.6	27.2 ± 4.6	0.20
Serum albumin (g/dl)	3.5 ± 0.5	3.4 ± 0.5	0.04
Serum creatinine (mg/dl)	7.2 ± 3.1	7.4 ± 3.3	0.02
Serum ferritin (ng/ml)	339 (384)	372 (403)	0.01
Serum total iron binding capacity (mg/dl)	241.8 ± 51.8	235 ± 52.5	<0.001
Serum bicarbonate (mg/dl)	25.4 ± 3.1	25.0 ± 3.3	0.001
Serum phosphorus (mg/dl)	5.1 ± 1.3	5.3 ± 1.4	0.005
Serum calcium (mg/dl)	9.1 ± 0.7	9.0 ± 0.8	0.05
Blood hemoglobin (g/dl)	12.2 ± 1.4	12.1 ± 1.6	0.002
White blood cell count ($\times 10^3$ per μl)	7.9 ± 2.7	8.0 ± 3.2	0.08
Lymphocyte (% of total WBC count)	18.8 ± 7.0	18.1 ± 7.5	0.01

Data represent 13-week average measurements during the baseline (first calendar) quarter.

into the cohort. The study cohort of 2798 diabetic PD patients was identified after excluding individuals without diabetes (n = 6439) and those diabetics with no data on A1c (n = 1291). Of the 2798 eligible patients who formed the study cohort, 620 patients were prevalent in the first quarter (July 1, 2001 through September 30, 2001) and 2178 accumulated over the subsequent 19 quarters. The median follow-up time was 640 days.

Table 1 shows baseline demographic, clinical, and laboratory characteristics of the diabetic patients with and without data on A1c (n = 2798 and 1291, respectively). Individuals with missing data were less likely to have diabetes as a cause of ESRD, had longer dialysis vintage, were more likely to have Medicare as a primary insurance, and had lower total iron binding capacity. The differences in many of the other characteristics, although statistically significant because of a large sample size, were small (Table 1).

Table 2 shows baseline demographic, clinical, and laboratory characteristics of the studied PD patients according to seven *a priori* categories based on baseline A1c—higher A1C levels were associated with lower serum creatinine, albumin, and ferritin levels.

Figure 1A (and Supplemental Table e1) shows unadjusted and adjusted death hazard ratios for groups based up baseline A1c. Case-mix and MICS adjusted all-cause death hazard ratio (HR) and 95% confidence interval (95% CI) for A1c increments of 7.0 to 7.9%, 8.0 to 8.9%, 9.0 to 9.9%, and ≥10%, compared with 6.0 to 6.9% (reference), were 1.13 (0.97 to 1.32), 1.05 (0.88 to 1.27), 1.06 (0.84 to 1.34), and 1.48 (1.18 to 1.86). However, time-averaged A1c ≥8% was associated with a higher risk for all-cause mortality (Figure 1B and Supplemental Table e2).

Hemoglobin level (>11.0 or ≤11.0 g/dl) was identified as a significant modifier of the baseline A1c-mortality association (P value for interaction term, 0.004). In 2264 or 81% of diabetic PD patients, blood hemoglobin was >11.0 g/dl (28). Supplemental Figure e2, A and B, and Supplemental Tables e3 and e4 show the same analyses as in Figure 1A for nonanemic (upper panel) and anemic (lower panel) PD patients. Among nonanemic patients, A1c ≥10% was associated with 53% higher all-cause mortality (reference: A1c 6.0 to 6.9%; HR, 1.53 (1.18 to 1.98)). However, there was no association of A1c with outcome in patients with Hb ≤11.0 g/dl.

Subsequent subgroup analyses were performed to examine the hazard ratios for all-cause mortality for patients with baseline A1c ≥7% among relevant demographic, clinical, and laboratory categories of PD patients (Figure 2). In the entire PD population, the HR for all-cause mortality in patients with baseline A1c ≥7% was 1.10 (0.98 to 1.23); however, in Caucasians (interaction term for Caucasians: P = 0.063), men (interaction term for gender: P = 0.075), and patients with albumin >3.8 g/dl (interaction term for albumin groups: P = 0.24), the death HRs of baseline A1c ≥7% were 1.22 (1.05 to 1.42), 1.26 (1.08 to 1.48), and 1.48

Table 2. Demographic, clinical, and laboratory values in 2798 PD patients and according to the categories of A1c

Variables	A1c Categories							*P* for trend
	<5.0% (*n* = 102)	5.0 to 5.9% (*n* = 479)	6.0 to 6.9% (*n* = 747)	7.0 to 7.9 (*n* = 649)	8.0 to 8.9% (*n* = 420)	9.0 to 9.9% (*n* = 217)	≥10% (*n* = 184)	
Age (years)	57 ± 12	61 ± 12	60 ± 13	58 ± 13	56 ± 13	54 ± 13	54 ± 13	<0.001
Gender (% women)	43.1	43.8	44.2	41.6	45.2	42.4	48.4	0.54
Race/ethnicity (%)								0.41
Caucasian	47.1	54.2	54.5	51.8	53.2	48.9	50.5	
African American	27.5	20.7	17.8	20.8	17.4	19.4	22.3	
Hispanic	13.7	15.3	14.8	14.8	16.2	22.1	19.0	
Vintage (dialysis time, %)								<0.001
<6 months	63	71	71	68	74	76	71	
6 to 24 months	19	16	15	18	16	14	19	
2 to 5 years	14	11	12	12	8	8	8	
>5 years	4	2	2	2	2	2	2	
Primary insurance								
Medicare (%)	53.4	61.0	61.7	58.3	53.6	56.0	57.5	0.20
Body mass index (kg/m^2)	26.4 ± 4.5	26.7 ± 4.5	27.1 ± 4.4	27.0 ± 5.2	27.2 ± 4.7	26.6 ± 4.3	26.5 ± 4.1	0.42
Serum albumin (g/dl)	3.55 ± 0.55	3.46 ± 0.45	3.46 ± 0.47	3.46 ± 0.45	3.44 ± 0.43	3.40 ± 0.45	3.37 ± 0.45	0.02
Serum creatinine (mg/dl)	8.3 ± 3.3	7.4 ± 3.2	7.2 ± 3.1	7.0 ± 2.9	6.9 ± 2.9	6.8 ± 2.8	6.4 ± 2.8	<0.001
Serum ferritin (ng/ml)	399 ± 361	383 ± 371	366 ± 500	308 ± 328	288 ± 279	294 ± 320	352 ± 327	<0.001
Serum total iron binding capacity (mg/dl)	233 ± 62	237 ± 56	246 ± 53	241 ± 50	243 ± 49	240 ± 45	236 ± 50	0.02
Serum bicarbonate (meq/l)	24.2 ± 3.2	25.2 ± 3.1	25.4 ± 3.0	25.5 ± 3.0	25.8 ± 3.1	25.8 ± 3.0	25.3 ± 3.1	<0.001
Serum phosphorus (mg/dl)	5.5 ± 1.5	5.1 ± 1.2	5.1 ± 1.3	5.1 ± 1.4	5.1 ± 1.3	5.1 ± 1.3	5.2 ± 1.2	0.05
Serum calcium (mg/dl)	9.0 ± 0.9	9.1 ± 0.8	9.1 ± 0.7	9.1 ± 0.7	9.1 ± 0.7	9.0 ± 0.8	8.9 ± 0.7	0.01
Blood hemoglobin (g/dl)	11.8 ± 1.7	12.1 ± 1.4	12.2 ± 1.4	12.4 ± 1.4	12.4 ± 1.4	12.4 ± 1.4	12.3 ± 1.6	<0.001
White blood cell count ($\times 10^3$ per μl)	7.1 ± 2.1	7.5 ± 2.4	7.8 ± 2.7	8.1 ± 2.4	8.0 ± 2.3	8.4 ± 4.8	7.8 ± 2.0	<0.001
Lymphocyte (% of white blood cell count)	18.3 ± 7.0	18.4 ± 6.8	19.0 ± 7.5	18.6 ± 6.8	18.8 ± 6.6	19.4 ± 6.9	19.4 ± 6.7	0.48

Data represent 13-week average measurements during the baseline (first calendar) quarter.

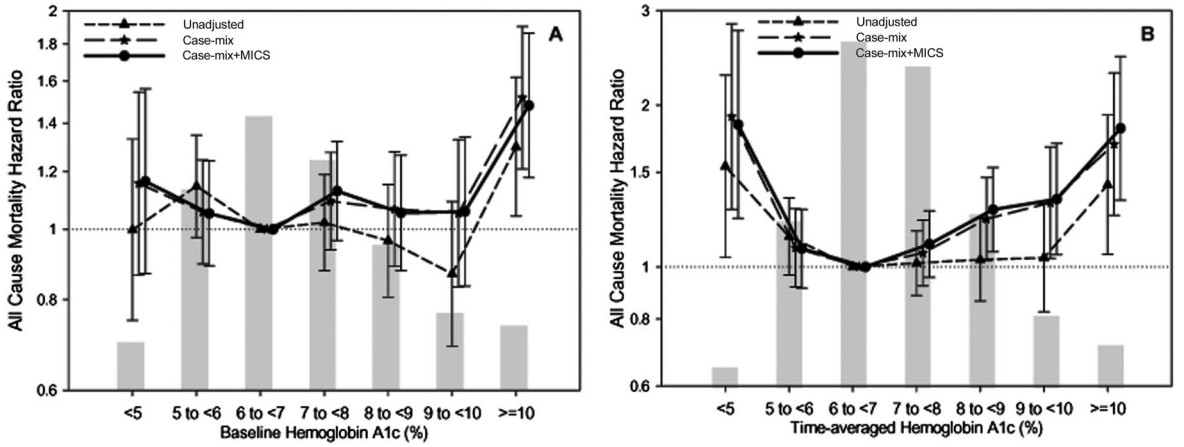

Figure 1. | **HRs of all-cause mortality of the entire range of A1C in 2798 PD patients using standard Cox (A) and time-averaged (B) models.** Case-mix model is adjusted for age, sex, race/ethnicity, pre-existing comorbid states, tobacco smoking, dialysis vintage, primary insurance, and marital status. Malnutrition-inflammation complex syndrome (MICS)–adjusted model includes all of the case-mix covariates as well as body mass index and 10 laboratory variables of nutrition and inflammation.

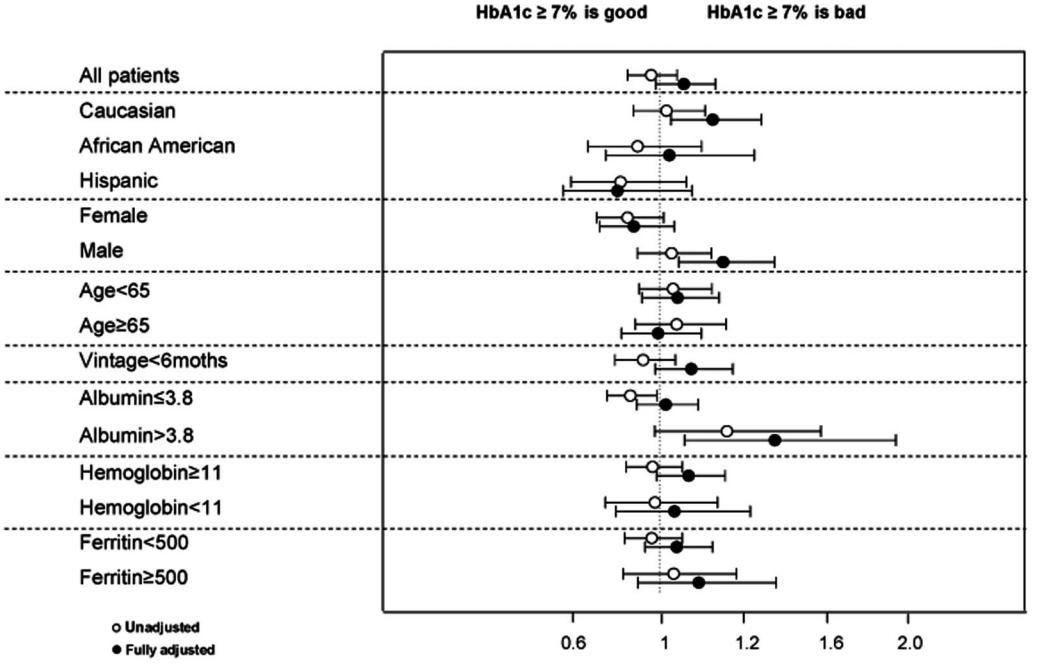

Figure 2. | **HRs of all-cause mortality for the dichotomized A1C >7% in different subgroups of 2798 PD patients.** Adjusted model is controlled for age, sex, race/ethnicity, pre-existing comorbid states, tobacco smoking, dialysis vintage, primary insurance, marital status, body mass index, and 10 laboratory variables of nutrition and inflammation.

(1.10 to 1.98), respectively. Additional analysis for the predictive value of baseline A1c ≥7% was performed by stratifying the group based on what the underlying cause of ESRD was (diabetes *versus* other). The HR for all-cause mortality in case-mix and MICS model was 1.06 (0.94 to 1.20) for patients with diabetes as the cause of ESRD and 1.27 (0.89 to 1.81) in those with another underlying cause of ESRD.

We repeated the analyses using cardiovascular death as outcome. Supplemental Figure e3 and Supplemental Table e5 show unadjusted and adjusted hazard ratios according to the baseline A1c values. We did not find any difference in mortality risk between different baseline A1c groups. We also performed subgroup analyses using cardiovascular death outcome to examine the existence of interaction between anemia and A1c. These A1c-mortality associations were not significantly different across anemia subgroups (Figure 2), and the inclusion of the interaction term was nonsignificant ($P = 0.16$). Subsequent subgroup analyses were performed to examine the statistical interaction by estimating the hazard ratios for cardiovascular mortality for the baseline A1c ≥7% among relevant demographic,

clinical, and laboratory categories of PD patients (Supplemental Figure e3). In this analysis we found that baseline A1c ≥7% was associated with significantly higher risk of cardiovascular mortality only in patients with albumin higher than 3.8 g/dl, but not among Caucasians or men.

We found moderate, significant correlation between serum glucose and A1C level ($r = 0.51$). In the analysis using blood glucose as a predictor variable, we found similar results as seen with A1c. Case-mix and MICS adjusted all-cause death HR for baseline blood glucose 100 to 149, 150 to 199, 200 to 249, 250 to 299, and ≥300 mg/dl, compared with 60 to 99 mg/dl (reference), were 1.00 (0.76 to 1.32), 0.98 (0.74 to 1.29), 1.14 (0.86 to 1.51), 1.05 (0.77 to 1.44), and 1.33 (0.97 to 1.83), respectively (Figure 3A and Supplemental Table e6). Time-averaged blood glucose ≥300 mg/dl was associated with a significantly higher risk for all-cause mortality 2.10 (1.37 to 3.20) (reference, glucose, 60 to 99 mg/dl) (Figure 3B and Supplemental Table e7).

Discussion

In this large-scale contemporary cohort of 2798 PD patients, we report that only a time-averaged A1c ≥8% or serum glucose ≥300 mg/dl appears to be associated with higher all-cause mortality. This association was particularly robust in diabetic PD patients with hemoglobin ≥11 g/dl. Subgroup analyses showed that the A1c threshold for higher all-cause mortality was lower in Caucasians, men, and patients with albumin level higher than 3.8 g/dl (A1C ≥7%). These findings may have important clinical implications, especially because they imply moderate hyperglycemia may not be a risk factor for death for this population. This is particularly relevant for patients in whom the dialysis therapy itself imposes an obligatory glucose burden, over and above the dietary intakes.

The literature on the relationship between glycemic control and survival in the chronic kidney disease (CKD) population is somewhat limited. In a cohort of 840 nondiabetic patients with moderate CKD who participated in the Modification of Diet in Renal Disease trial, A1c was a predictor of all-cause mortality (29). However, a study using data from patients treated in units owned by Fresenius was unable to demonstrate any association between A1c and 1-year survival in 24,875 hemodialysis patients (11). This contrasts with several other observational studies: Wu *et al.* studied 137 hemodialysis patients with type 2 diabetes and reported that the cumulative survival was lower in the group with poor glycemic control (16). Similarly, we have previously shown that higher A1c is associated with increased death risk in patients treated with hemodialysis (21). Recently, Williams *et al.* reported a higher risk for death only in type 2 diabetic hemodialysis patients with A1c levels >11% (20). The findings reported herein are consistent with these previous findings in hemodialysis patients and with the only other study that has examined the relationship between glycemic control and outcome in PD patients, which also found poor glycemic control to be associated with higher death risk in 101 PD patients (24).

There are several possible mechanisms that might explain the relationship between glycemic control and survival of PD patients. Poor glycemic control might result directly in macrovascular complications, possibly secondary to the generation of advanced glycation end products (AGEs), and hence shorten survival of these patients. However, higher AGE levels in 312 hemodialysis patients were found to be paradoxically associated with better survival (30). Whether the benefit of high serum AGEs in these types of observational studies is an epiphenomenon or reflects a better nutritional status needs further study. Furthermore, comorbid conditions might make the glycemic control unsatisfactory and the higher risk for death may be secondary to the comorbid conditions rather than the poor glycemic control itself. Finally, in PD patients, higher A1c levels may indicate greater cumulative peritoneal glucose exposure with its attendant damage to the peritoneal membrane. This damage to peritoneal membrane is similar, although not identical, to the systemic microvascular disease seen in diabetics and is associated with peritoneal neo-vascularization. This, in turns, leads to a faster perito-

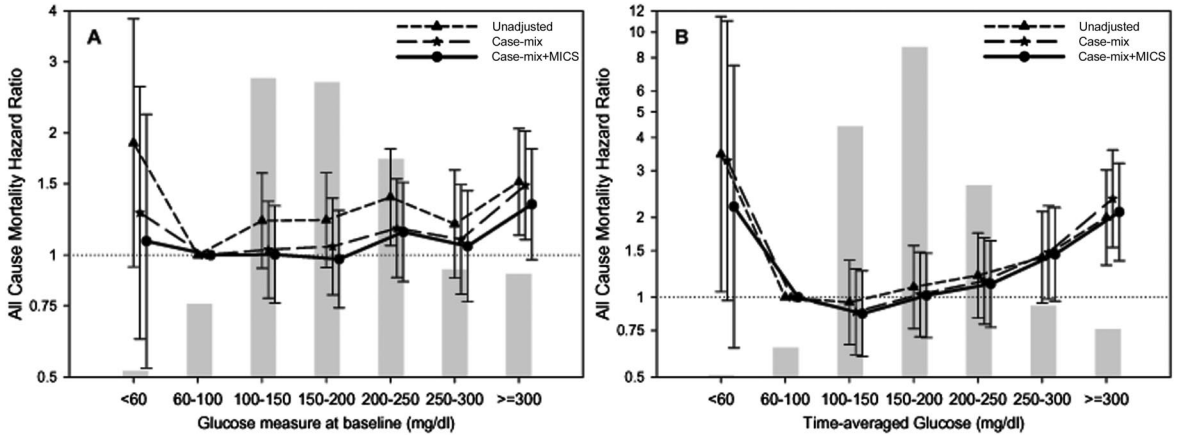

Figure 3. | HRs of all-cause mortality of serum glucose in 2575 diabetic PD patients using standard Cox (A) and time-averaged (B) models. Case-mix model is adjusted for age, sex, race/ethnicity, pre-existing comorbid states, tobacco smoking, dialysis vintage, primary insurance, and marital status. Malnutrition-inflammation complex syndrome (MICS)–adjusted model includes all of the case-mix covariates as well as body mass index and 10 laboratory variables of nutrition and inflammation.

neal transport, reduced ultrafiltration capacity, and consequent volume overload and has been associated with a higher risk for death (31,32).

An interesting finding in our study was the significant interaction between anemia and serum albumin and mortality predictability of A1c. Hence, the association between high A1c and all-cause mortality was particularly robust in individuals with higher hemoglobin (>11 g/dl), and was evident at lower A1c levels in those with higher serum albumin (≥3.8 g/dl). These findings may indicate the possible interaction of factors related to protein-energy wasting, inflammation, and anemia with indices of glycemic control.

The information on comorbidity in our study was limited to that obtained from Medical Evidence Form 2728, a form in which comorbid conditions are significantly under-reported (27). Moreover, we did not have any data available on the medications, if any, to treat diabetes mellitus and/or their doses, nor did we study patient adherence with therapy. Furthermore, the required dose of these medications can be confounded by the residual renal function and its deterioration over time (18). Another potential limitation is lack of explicit laboratory markers of inflammation such as C-reactive protein. However, we used data on serum albumin, ferritin and total iron binding capacity, blood white blood cell count, lymphocyte percentage, and hemoglobin, which have significant associations with inflammation in dialysis patients (26). An additional limitation of our study is that we had to exclude 1291 patients in whom no A1c data were available. We also did not have data on PD modality (continuous ambulatory or automated PD), dialysis prescription, peritoneal transport rate, or adequacy of dialysis.

Conclusions

In conclusion, poor glycemic control (A1c ≥8% or serum glucose ≥300 mg/dl) appears to be associated with decreased survival in PD patients. Our study suggests that moderate hyperglycemia may not increase the risk for all-cause or cardiovascular mortality of diabetic PD patients, except in certain subgroups (Caucasians, men, and those with serum albumin >3.8 g/dl). However, mortality is only one measure of the deleterious impact of poor glycemic control. Other potential benefits of glycemic control including slowing the rate of progression of microvascular disease and rate of loss of residual renal function are possible, which have not been studied herein. Clinical trials are needed to better define the target A1c levels in different subgroups of diabetic PD patients.

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Disclosures

None.

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