Developing an HbA$_{1c}$-Based Equation to Estimate Blood Glucose in Maintenance Hemodialysis Patients

JUNICHI HOSHINO, MD, MPH$^{1,2,3}$
MIKLOS Z. MOLNAR, MD, PHD$^{1,4,5}$
KUNIHIRO YAMAGATA, MD, PHD$^6$
YOSHIFUMI UBARA, MD, PHD$^7$
KENMEI TAKAICHI, MD, PHD$^3$
CSABA P. KOVESDY, MD$^{7,8}$
KAMYAR KALANTAR-ZADEH, MD, MPH$^1,2,9$

OBJECTIVE—Hemoglobin A$_{1c}$ (HbA$_{1c}$) has been widely used as a clinically important assessment tool for outcome analyses related to glycemic control. However, because of special conditions in dialysis patients, including the uremic milieu, there is no HbA$_{1c}$ blood glucose (BG) equation specific for patients on dialysis. In this study, we sought to develop HbA$_{1c}$-BG equation models for hemodialysis patients.

RESEARCH DESIGN AND METHODS—We examined associations between HbA$_{1c}$ and random serum BG over time in a contemporary cohort of diabetic patients with hemodialysis treated in DaVita dialysis clinics. We identified 11,986 patients (63 ± 12 years old and 49% male) with 67,764 paired measurements of HbA$_{1c}$ and BG over the course of 5 years (2001–2006). Bootstrapping method was used to estimate average BG and corresponding HbA$_{1c}$ levels. The association was adjusted by patient factors using linear regression.

RESULTS—Linear regression analyses yielded the following three regression equations: BG = 59.2 + 29.4 × HbA$_{1c}$ − 20.8 × Alb (R$^2$ = 0.483); BG = 104.8 + 29.7 × HbA$_{1c}$ − 18.4 × Alb + 4.7 × Hb (R$^2$ = 0.486); and BG = 82.9 + 30.7 × HbA$_{1c}$ − 16.5 × Alb − 5.4 × Hb + 0.3 × age + race (R$^2$ = 0.491). All our models showed stronger association than previous equation models (R$^2$ = 0.468 in the Diabetes Control and Complications Trial and A1c-Derived Average Glucose equations).

CONCLUSIONS—The association between HbA$_{1c}$ and BG in hemodialysis patients is different than that of patients with normal kidney function. Our analysis suggests that equations including serum albumin or hemoglobin are better for hemodialysis patients.

Hemoglobin A$_{1c}$ (HbA$_{1c}$) has been widely used as a clinically important assessment tool for outcome analyses related to glycemic control. Numerous studies have documented that HbA$_{1c}$ is highly correlated with a patient’s directly measured blood glucose (BG) levels averaged over time. There are some equations showing a good correlation between HbA$_{1c}$ and average BG (AG) levels (the Diabetes Control and Complications Trial [DCCT] formula: AG = 35.6 × HbA$_{1c}$ − 77.3; and the A1c-Derived Average Glucose [ADAG] study: AG = 28.7 × HbA$_{1c}$ − 46.7) (1,2). The recommendation of ADAG group implies that HbA$_{1c}$ is a reliable substitute for mean BG and, except for analytic variation, the only important factor determining HbA$_{1c}$ is the preceding mean BG.

Glycation rate is determined by temperature, pH, hemoglobin (Hb) concentration, BG concentration, and length of exposure to glucose (3). Because the Hb concentration and pH of dialysis patients can be significantly abnormal, the correlation between HbA$_{1c}$ and AG levels in dialysis patients is considered different from that of normal patients. Furthermore, shortened erythrocyte life span and accelerated erythropoesis because of routine use of erythropoietin could affect HbA$_{1c}$ levels in dialysis patients. HbA$_{1c}$ was found to underestimate glucose measurements in diabetic patients on dialysis compared with glycated albumin (4–6). Unfortunately, there has been no HbA$_{1c}$-AG equation specific for hemodialysis (HD) patients, despite the increasing number of diabetic patients on dialysis. In this study, we sought to develop HbA$_{1c}$-AG equation models for HD patients.

RESEARCH DESIGN AND METHODS

Database creation

The data were obtained from DaVita, the second largest dialysis care provider in the United States, with ~500 dialysis centers and 40,000 patients across the country. The creation of the national DaVita dialysis patient cohort has been described previously (7–12). A 60-month prevalent cohort (July 2001–June 2006) of DaVita maintenance HD patients was studied. Demographic data and details of medical history were collected, with information on age, gender, race, and presence of diabetes. The study conformed to Declaration of Helsinki and Good Clinical Practice Guidelines.

Laboratory measures

Blood samples were drawn using uniform techniques in all the DaVita dialysis clinics and were transported to the DaVita Laboratory in Deland, Florida, typically within 24 h. All laboratory values were
measured by automated and standardized methods in the DaVita Laboratory. Most laboratory values were measured monthly, including serum urea nitrogen, creatinine, serum albumin (Alb), BG, calcium, phosphate, and bicarbonate. Hb was measured at least monthly in essentially all patients. HbA1c was measured at least quarterly. All these variables were measured before HD, with the exception of postdialysis serum urea nitrogen.

**Statistical methods**

Data were summarized using proportions, means (±SD) as appropriate. Categorical variables were analyzed with the χ² test or Fisher exact test as appropriate, and continuous variables were compared using t test, Mann-Whitney U test, Kruskal-Wallis H test, or ANOVA, as appropriate. Patients receiving dialysis for <90 days, those without diabetes, those with missing values of Alb, glucose, Hb, or HbA1c, and those who were tested for values less than three times were excluded from this analysis. Laboratory values in this data set were refined using limits as follows: albumin, 1.0–5.0 g/dL; BG, 30–500 mg/dL; Hb, 3–20 g/dL; and HbA1c 1–12 mg/dL, respectively. All repeated measurements of every relevant variable for each patient within each calendar quarter or 13 weeks were averaged to obtain one quarterly mean value for that given variable. Averaged HbA1c values and BG values were compared using regression models with and without adjustment for Hb, Alb, patient age, gender, or race or ethnicity. In addition to linear models, quadratic, root, log, and exponential regression models also were applied to estimate the relationship between averaged HbA1c and BG. Bootstrapping method also was used to estimate AG and corresponding HbA1c for more reliable estimates. One thousand resampling procedures with replacement were selected to approximate the sampling distribution of statistics of interest. R² and root mean square error were used to compare the correlations between the simple and multivariate regression models. After identifying the best models, we applied them to gender-based and race-based categories to compare the accuracy of the models in each subgroup. The DCCT and the ADAG formulas were used as control formulas.

**RESULTS**

**Equation model creation**

Baseline characteristics are shown in Table 1. The mean age was 63 ±12 years, which was slightly higher in Asians (66 ±11 years) than in African Americans (62 ±12 years). Most patients were non-Hispanic white (33%), African American (34%), and Hispanic (21%), with relatively few Asian and other races, including Pacific Islander and Native Americans. The mean dialysis vintage was 2.9 ± 2.5 years. Eighty percent of patients in this study had type 2 diabetes mellitus. Most characteristics were similar in males and females.

The relationship between the average HbA1c and the AG (n = 11,986) is shown in Fig. 1 and Table 2. The scatter plot suggests that there is a linear relationship between HbA1c and BG, which is the same as the relationship previously reported in nondialysis populations (1,2). Rohlfing et al. (1) also reported that afternoon and evening BG showed higher correlations with HbA1c than did the morning values. Our analysis also showed that the relationship between HbA1c and BG among patients in the afternoon or evening shift (R² = 0.494) was better than during the morning (R² = 0.477) (see Supplementary Fig. 1). Next, we compared several equation models with and without adjustment for confounding factors. As shown in Table 2, the equation improved if Alb was added (R² = 0.468–0.483) to the model. The R² gradually improved after adding age or race variables, or both, although the degree of improvement was not large. We could not find any improvement after adding gender.

**Table 1—Baseline patients’ characteristics (n = 11,986)**

| Characteristics                  | n or mean ± SD | Caucasian | African American | Hispanic | Asian | Other | P† |
|----------------------------------|----------------|-----------|------------------|----------|-------|-------|----|
| Total                            | 11,986 100     | 3,963 33  | 4,093 34         | 2,521 21 | 402 3 | 1,007 8 | <0.001 |
| Age (years)                      | 63 ± 12        | 65 ± 13   | 62 ± 12          | 62 ± 11  | 66 ± 11 | 63 ± 11 | <0.001 |
| Gender                           |                |           |                  |          |       |       |    |
| Female                           | 6,098 51       | 1,824 46  | 2,316 57         | 1,252 50 | 211 52 | 495 49 | <0.001 |
| Male                             | 5,888 49       | 2,139 54  | 1,777 43         | 1,269 50 | 191 48 | 512 51 |    |
| Type of diabetes                 |                |           |                  |          |       |       |    |
| Type 1                           | 2,360 20       | 839 21    | 855 21           | 439 17   | 66 16 | 161 17 | <0.001 |
| Type 2                           | 9,626 80       | 3,124 79  | 3,238 79         | 2,082 83 | 336 84 | 846 83 |    |
| Dialysis vintage (years)         | 2.8 ± 2.4      | 2.3 ± 2.0 | 3.2 ± 2.7        | 2.8 ± 2.4 | 2.6 ± 2.2 | 3.2 ± 2.7 | <0.001 |
| Laboratory findings              |                |           |                  |          |       |       |    |
| Total protein (g/dL)             | 6.9 ± 0.5      | 6.6 ± 0.5 | 7.1 ± 0.5        | 6.9 ± 0.5 | 7.0 ± 0.5 | 6.9 ± 0.5 | <0.001 |
| Albumin (g/dL)                   | 3.7 ± 0.3      | 3.7 ± 0.3 | 3.7 ± 0.3        | 3.7 ± 0.3 | 3.8 ± 0.3 | 3.7 ± 0.3 | <0.001 |
| Hb (g/dL)                        | 12.0 ± 0.7     | 12.0 ± 0.7 | 12.0 ± 0.7      | 12.1 ± 0.7 | 12.0 ± 0.6 | 12.0 ± 0.7 | <0.001 |
| Glucose (mg/dL)                  | 175 ± 54       | 179 ± 54  | 166 ± 53         | 179 ± 55 | 178 ± 51 | 175 ± 53 | <0.001 |
| HbA1c (%)                        | 6.6 ± 1.3      | 6.5 ± 1.2 | 6.5 ± 1.3        | 6.7 ± 1.3 | 6.4 ± 1.1 | 6.6 ± 1.3 | <0.001 |
| Dialysis information             |                |           |                  |          |       |       |    |
| Dialysis time (h)                | 3.5 ± 0.8      | 3.5 ± 0.8 | 3.6 ± 0.8        | 3.5 ± 0.7 | 3.1 ± 0.9 | 3.4 ± 0.8 | <0.001 |
| Kt/V*                            | 1.53 ± 0.33    | 1.52 ± 0.33 | 1.48 ± 0.30     | 1.58 ± 0.33 | 1.67 ± 0.38 | 1.60 ± 0.36 | <0.001 |

*Kt/V* was estimated by Daugirdas II equation for single pool. †P derived from ANOVA and Pearson χ² tests.
A new HbA1c-glucose equation for hemodialysis patients

Blood glucose estimation

Next, we compared AG and estimated BG using our models and previously reported equations (ADAG and DCCT models established for nondialysis populations). Compared with previous equations (ADAG and DCCT models established for nondialysis populations; adjusted $R^2 = 0.468$), all of our models showed better correlation (adjusted $R^2 = 0.483, 0.486,$ and 0.491) (Fig. 2) in HD patients. In addition, the glucose levels estimated by the DCCT and ADAG models were similar to those of our models if patients had Alb level of 4.0 mg/dL and if HbA1c was $>9.0\%$. However, the estimated glucose levels by the previous models were lower if patients had lower Alb levels or lower HbA1c levels (Table 3).

Utility of the model in race- and gender-based subgroups

Because our models suggested that race also was an independent covariate in the HbA1c–AG equation model, we checked correlations separately in each race group. Moreover, because the number of Asians was relatively small ($n = 402; 3\%$ of the study population), it was essential to check the utility of the equation in this group. As shown in Table 4, the adjusted $R^2$ between the AG and the estimated BG using our model 3 and model 8 in non-Hispanic whites were 0.470 and 0.479 in males and 0.466 and 0.472 in females. There was a higher correlation in African Americans (adjusted $R^2 = 0.530$ and 0.538 in males and 0.531 and 0.538 in females) and Hispanics (adjusted $R^2 = 0.498$ and 0.511 in males and 0.478 and 0.488 in females) compared with non-Hispanic whites, but a lower association among Asians (adjusted $R^2 = 0.385$ and 0.387 in males and 0.400 and 0.414 in females). These data suggested that this model may work especially well in African American and Hispanic populations.

CONCLUSIONS—In this large-scale cohort of 11,986 diabetic HD patients, we reported new HbA1c and BG equation models that are at least as good or better than previous equations. To the best of our knowledge, this is the first model for HD patients showing better correlation than the previous standard formulas that were developed for nondialysis patients. Interestingly, the slope of the glucose variable in our model was almost identical to that in the ADAG model. HbA1c has been considered to underestimate glucose measurements in diabetic patients on HD compared with glycated albumin (4–6).

Using the ADAG and DCCT model, the estimated glucose levels tended to be lower than the AG (Table 3).

Although previous observational studies have yielded inconsistent results regarding the association between glycemic control and outcomes in diabetic HD patients, our recent study suggested that after adjusting for potential confounders, higher HbA1c values were associated with higher death risk in patients on maintenance HD (13,14). Therefore, HbA1c is considered an important clinical marker for glycemic control. There have been several other measurements that represent glycemic levels for patients with chronic kidney disease, such as glycated albumin (15) and 1,5-anhydro-d-glucitol (16). However, an ideal indicator for glycemic control in dialysis patients has not been agreed on yet. All of the proposed markers have advantages and disadvantages. For example, the accelerated destruction of erythrocytes may reduce the half-life of HbA1c; however, glycoalbumin may be affected by an accelerated turnover of Alb with proteinuria, which is commonly observed in patients with end-stage renal disease. Nevertheless, HbA1c still has been one of the most widely used glycemic control indicators for diabetic patients with and without kidney disease. A clear understanding of the relationship between AG and HbA1c is necessary for setting appropriate BG goals for achieving specific HbA1c targets in dialysis patients. Our model may be useful to convert daily AG to target HbA1c values and vice versa. Because most HD patients are considered as having similar Hb levels, which is recommended by clinical guidelines, model 3 (AG = 59.2 + 29.4 × HbA1c − 20.8 × Alb) may be the most simple and practical equation for daily use.

Rambod et al. (17) reported that low HbA1c levels could be considered a surrogate marker of protein-energy wasting, which is a well-known predictor of mortality in dialysis patients. Similarly, we previously reported that Alb levels were associated with HbA1c levels (14). As shown in Table 3, the estimated glucose levels in this study were higher by $\sim 20$ mg/dL if Alb levels decreased from 4.0 to 3.0 g/dL. Patients on dialysis have a higher prevalence of hypercatabolism compared with the general population, which could affect Alb levels (18). Our results supported a simple linear relationship between mean glucose and HbA1c levels in a clinically relevant range of glycemia that was reported by Nathan et al.

Figure 1—Relationship between mean HbA1c and mean BG in each patient on HD ($n = 11,986$). Comparison between average HbA1c and AG levels in each patient ($n = 11,986$). The $R^2$ in the fitted line was 0.468.
methods were used to estimate averaged BG and averaged HbA1c.

However, it also revealed the importance of taking into account the Alb level in the equation between HbA1c and AG for dialysis patients, especially because Alb values in dialysis patients are usually lower than they are in the general population. In addition, our analysis revealed that careful interpretation of HbA1c is needed if patients had malnutrition and anemia, known as MIA syndrome. In these patients, equation models including Alb and Hb (model 4 or complex model) may be useful to estimate their average glucose levels.

It was reported that postprandial BG was associated with survival in HD patients (19) and there was no difference for mean amplitude of glycemic excursion between the day on and day off HD by an analysis of continuous glucose monitoring (20). In addition, as shown in the Supplementary Fig. 1, our data showed that afternoon and evening BG measurements, not fasting BG, had better correlation with their HbA1c levels, which was consistent with the results for patients without end-stage renal disease reported by Rohlfing et al. (1). These findings may imply that initiation of HD may be best after BG measurement. If patients start dialysis with fasting, then postprandial glucose measurement should be added. Also if subjects use maintained antidiabetes agents, BG measurement during HD should be added, because glucose levels decrease with initiation of HD when using these agents (20).

There are several limitations in this study. First, our data were not based on continuous daily BG monitoring. However, the large number of random BG samples available to us and the bootstrapping technique may minimize this problem. Furthermore, our equations obtained by bootstrapping showed similar results with the estimation obtained by using time-averaged values, suggesting robustness of our models. Second, the proportion of the

Table 2—Relationships between the AG and the average HbA1c.

| Model | Covariates | Mean* | Bootstrapping† |
|-------|------------|-------|---------------|
|       |            | Adjusted $R^2$ | RMSE | Adjusted $R^2$ | RMSE |
| 1     | $AG = -18.6 + 29.4 \times HbA1c$ | 0.468 | 39.140 | 0.468 | 39.151 |
| 2     | $AG = 59.6 + 29.8 \times HbA1c - 6.7 \times Hb$ | 0.475 | 38.871 | 0.475 | 38.879 |
| 3     | $AG = 59.2 + 29.4 \times HbA1c - 20.8 \times Alb$ | 0.483 | 38.578 | 0.483 | 38.583 |
| 4     | $AG = 104.8 + 29.7 \times HbA1c - 18.4 \times Alb - 4.7 \times Hb$ | 0.486 | 38.453 | 0.486 | 38.457 |
| 5     | $AG = 25.1 + 30.4 \times HbA1c - 19.5 \times Alb + 0.4 \times age$ | 0.489 | 38.358 | 0.489 | 38.365 |
| 6     | $AG = 25.2 + 30.4 \times HbA1c - 19.6 \times Alb + 0.4 \times age + 0.9 \times gender$ | 0.489 | 38.357 | 0.489 | 38.364 |
| 7     | $AG = 32.3 + 30.3 \times HbA1c - 19.4 \times Alb + 0.3 \times age + race$ | 0.498 | 37.988 | 0.498 | 37.995 |
| 8     | $AG = 82.9 + 30.7 \times HbA1c - 16.5 \times Alb - 5.4 \times Hb + 0.3 \times age + race$ | 0.503 | 37.822 | 0.503 | 37.828 |
| 9     | $AG = 83.1 + 30.7 \times HbA1c - 16.6 \times Alb - 5.4 \times Hb + 0.3 \times age + 0.4 \times gender + race$ | 0.503 | 37.823 | 0.503 | 37.829 |
| 10    | $AG = 48.8 + 41.6 \times HbA1c - 0.8 \times (HbA1c)^2 - 16.8 \times Alb - 5.5 \times Hb + 0.3 \times age + race$ | 0.504 | 37.789 | 0.504 | 37.796 |

A, Asian; AA, African American; H, Hispanic; RMSE, root mean square error. *Mean BG and mean HbA1c were used to estimate correlations. †The bootstrapping methods were used to estimate averaged BG and averaged HbA1c.

Figure 2—Relationship between the AG and the estimated BG using estimation models. A: Relationship between AG and estimated BG using estimation models. Simple model 3: $AG = 59.2 + 29.4 \times HbA1c - 20.8 \times Alb$ ($R^2_{adj} = 0.483$). B: Simple model 4 was used: $AG = 104.8 + 29.7 \times HbA1c - 18.4 \times Alb - 4.7 \times Hb$ ($R^2_{adj} = 0.486$). C: Complex model 8: $AG = 82.9 + 30.7 \times HbA1c - 16.5 \times Alb - 5.4 \times Hb + 0.3 \times age + race + 3.8 \times A$ if Asian, 12.0 if African American, 3.3 if Hispanic ($R^2_{adj} = 0.491$). D: ADAG formula: $AG = 28.7 \times HbA1c - 46.7$ ($R^2_{adj} = 0.468$). $R^2_{adj}$, adjusted $R^2$. 

care.diabetesjournals.org
A new HbA1c-glucose equation for hemodialysis patients

Table 3—Estimated glucose levels at each HbA1c and Alb levels in the different models

| HbA1c (%) | Alb (mg/dL) | Estimated average glucose (mg/dL) |
|----------|-------------|----------------------------------|
|          | Model 3 (+Alb) | Model 4 (+Alb,Hb) | Model 8* (complex) | DCCT model | ADAG model |
| 5.0      | 4.0          | 123                 | 133                 | 134        | 101        | 97        |
| 5.0      | 3.5          | 133                 | 142                 | 143        |            |           |
| 5.0      | 3.0          | 144                 | 151                 | 151        |            |           |
| 6.0      | 4.0          | 152                 | 162                 | 165        | 136        | 126       |
| 6.0      | 3.5          | 163                 | 172                 | 173        |            |           |
| 6.0      | 3.0          | 173                 | 181                 | 182        |            |           |
| 7.0      | 4.0          | 182                 | 192                 | 196        | 172        | 154       |
| 7.0      | 3.5          | 192                 | 201                 | 204        |            |           |
| 7.0      | 3.0          | 203                 | 211                 | 212        |            |           |
| 8.0      | 4.0          | 211                 | 222                 | 227        | 208        | 183       |
| 8.0      | 3.5          | 222                 | 231                 | 235        |            |           |
| 8.0      | 3.0          | 232                 | 240                 | 243        |            |           |
| 9.0      | 4.0          | 241                 | 252                 | 257        | 243        | 212       |
| 9.0      | 3.5          | 251                 | 261                 | 265        |            |           |
| 9.0      | 3.0          | 261                 | 270                 | 274        |            |           |
| 10.0     | 4.0          | 270                 | 281                 | 288        | 279        | 240       |
| 10.0     | 3.5          | 280                 | 290                 | 296        |            |           |
| 10.0     | 3.0          | 291                 | 300                 | 304        |            |           |

Estimated glucose levels for whites with Hb 10.0 g/dL and age 60 years (+3.8 if Asian, −12.0 if African American, and −3.3 if Hispanic).

Asian population was small in our data set. Therefore, our model might not be representative enough for the HbA1c-AG association in Asians. In fact, compared with other races, the correlations in Asians were relatively weak. Third, our population consisted of patients with relatively early dialysis vintage. The association between HbA1c and glucose may be different in patients with longer vintage, because uremia and routine use of erythropoietin could affect HbA1c. Finally, our models may not represent patients on peritoneal dialysis (PD). Because they are exposed to a greater glucose charge present in dialysate solution, their glucose behavior may be different from that of patients on HD. In addition, lack of information on PD prescription would add more residual confounding. A large database consisting of PD patients is needed to develop HbA1c-AG equation models for patients on PD.

In conclusion, there was a predictable relationship between HbA1c and AG in HD patients. Our new models for HD patients showed better correlations between HbA1c and AG compared with previous models. The model including Alb is better-suited for HD patients because of the lower Alb levels in this population. Similar to reporting serum creatinine with the calculated glomerular filtration rate, HbA1c levels should provide patients and health care providers with a more useful index of day-to-day BG levels.

Acknowledgments—The study was supported by research grants to K.K.-Z. from the National Institute of Diabetes and Digestive and Kidney Diseases of the National Institutes of Health (R01 DK-078106), a research grant from DaVita Clinical Research, and a philanthropic grant from Mr. Harold Simmons. M.Z.M. received grants from the National Development Agency (KTIA-OTKA-UK 7K-HUMAN-MB08-A-81231) and from the Research and Technological Innovation Fund, and is the recipient of the Hungarian Eottos Scholarship (MOB/77-2/2012). K.K.-Z. is the medical director of DaVita Harbor-UCLA/AFMI in Long Beach, CA.

No potential conflicts of interest relevant to this article were reported.

J.H., M.Z.M., and K.K.-Z. researched the data. J.H. wrote the paper. M.Z.M., K.Y., Y.U., K.T., C.P.K., and K.K.-Z. reviewed the manuscript. K.Y., Y.U., K.T., and C.P.K. contributed to the discussion. K.K.-Z. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

The authors thank DaVita Clinical Research for providing the clinical data, analysis, and review for this research project.

References

1. Rohling CL, Wiedmeyer HM, Little RR, England JD, Tennill A, Goldstein DE. Defining the relationship between plasma glucose and HbA1c: analysis of glucose profiles and HbA1c in the Diabetes Control and Complications Trial. Diabetes Care 2002;25:275–278
2. Nathan DM, Kuenen J, Borg R, Zheng H, Schoenfeld D, Heine RJ; A1c-Derived Average Glucose Study Group. Translating the A1C assay into estimated average glucose values. Diabetes Care 2008;31:1473–1478
3. Kovesdy CP, Park JC, Kalantar-Zadeh K. Glycemic control and burnt-out diabetes in ESRD. Semin Dial 2010;23:148–156
4. Peacock TP, Shihabi ZK, Bleyer AJ, et al. Comparison of glycated albumin and hemoglobin A1c levels in diabetic subjects on hemodialysis. Kidney Int 2008;73:1062–1068
5. Nagayama H, Inaba M, Okabe R, et al. Glycated albumin as an improved indicator of glycemic control in hemodialysis patients with type 2 diabetes based on fasting plasma glucose and oral glucose tolerance test. Biomed Pharmacother 2009;63:236–240

6. Uzu T, Hatta T, Deji N, et al. Target for glycemic control in type 2 diabetic patients on hemodialysis: effects of anemia and erythropoietin injection on hemoglobin A1c. Ther Apher Dial 2009;13:89–94

7. Kalantar-Zadeh K, Streja E, Kovesdy CP, et al. The obesity paradox and mortality associated with surrogates of body size and muscle mass in patients receiving hemodialysis. Mayo Clin Proc 2010;85:991–1001

8. Kalantar-Zadeh K, Shah A, Duong U, Hechter RC, Dukkipati R, Kovesdy CP. Kidney bone disease and mortality in CKD: revisiting the role of vitamin D, calcimimetics, alkaline phosphatase, and minerals. Kidney Int Suppl 2010;117:S10–S21

9. Miller JE, Kovesdy CP, Norris KC, et al. Association of cumulatively low or high serum calcium levels with mortality in long-term hemodialysis patients. Am J Nephrol 2010;32:403–413

10. Miller JE, Kovesdy CP, Nissenson AR, et al. Association of hemodialysis treatment time and dose with mortality and the role of race and sex. Am J Kidney Dis 2010;55:100–112

11. Molnar MZ, Huang E, Hoshino J, et al. Association of pretransplant glycemic control with posttransplant outcomes in diabetic kidney transplant recipients. Diabetes Care 2011;34:2536–2541

12. Duong U, Mehrotra R, Molnar MZ, et al. Glycemic control and survival in peritoneal dialysis patients with diabetes mellitus. Clin J Am Soc Nephrol 2011;6:1041–1048

13. Kalantar-Zadeh K, Kopple JD, Regidor DL, et al. A1C and survival in maintenance hemodialysis patients. Diabetes Care 2007;30:1049–1055

14. Ricks J, Molnar MZ, Kovesdy CP, et al. Glycemic control and cardiovascular mortality in hemodialysis patients with diabetes: a 6-year cohort study. Diabetes 2012;61:708–715

15. Freedman BI, Andries L, Shihabi ZK, et al. Glycated albumin and risk of death and hospitalizations in diabetic dialysis patients. Clin J Am Soc Nephrol 2011;6:1635–1643

16. Kim WJ, Park C-Y, Lee K-B, et al. Serum 1,5-anhydroglucitol concentrations are a reliable index of glycemic control in type 2 diabetes with mild or moderate renal dysfunction. Diabetes Care 2012;35:281–286

17. Rambod M, Bross R, Zitterkoph J, et al. Association of Malnutrition-Inflammation Score with quality of life and mortality in hemodialysis patients: a 5-year prospective cohort study. Am J Kidney Dis 2009;53:298–309

18. Raj DS, Sun Y, Tzamaloukas AH. Hypercatabolism in dialysis patients. Curr Opin Nephrol Hypertens 2008;17:589–594

19. Shima K, Komatsu M, Kawahara K, Minaguchi J, Kawashima S. Stringent glycaemic control prolongs survival in diabetic patients with end-stage renal disease on haemodialysis. Nephrology (Carlton) 2010;15:632–638

20. Jung HS, Kim HI, Kim MJ, et al. Analysis of hemodialysis-associated hypoglycemia in patients with type 2 diabetes using a continuous glucose monitoring system. Diabetes Technol Ther 2010;12:801–807

Hoshino and Associates