Using Hemoglobin A1c to Derive Mean Blood Glucose in Peritoneal Dialysis Patients

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Key Words

Hemoglobin A1c · Blood glucose · Equation model · Glycemic control · Albumin · Hemoglobin · Peritoneal dialysis · Race

Abstract

Background: Although hemoglobin A1c (HbA1c) has been widely used as a clinical assessment tool for outcome analyses related to glycemic control, the relationship between HbA1c and average blood glucose (BG) specific to peritoneal dialysis (PD) patients with diabetes has not been characterized. We sought to develop HbA1c-BG equation models for PD patients. **Methods:** We examined associations between HbA1c and random serum BG values over time in a contemporary 5-year (2001–2006) cohort of DaVita PD patients with diabetes. We identified 850 patients (mean age: 58 ± 13 years, 56% male) with 4,566 paired measurements of HbA1c and BG. The bootstrapping method was

used to estimate average BG and corresponding HbA1c. **Results:** Linear regression analyses yielded the following HbA1c-BG equations: (1) BG (mg/dl) = 24.1 + 28.6 × HbA1c – 12.2 × albumin [adjusted R² (R²_{adj}) = 0.454], (2) BG = 55.3 + 28.8 × HbA1c – 10.2 × albumin – 3.3 × Hb (R²_{adj} = 0.457), and (3) BG = 69.5 + 28.7 × HbA1c – 10.1 × albumin – 3.7 × Hb – 0.1 × age + race/ethnicity (–10.1 African Americans, –5.4 other race/ethnicities; R²_{adj} = 0.457). All models showed greater explanatory power of BG variation than previously established HbA1c-BG equation models defined within non-PD cohorts [R²_{adj} = 0.446 for both the Diabetes Control and Complications Trial (DCCT) and the A1c-Derived Average Glucose (ADAG) equations]. **Conclusions:** The association between HbA1c and BG in PD patients is different than that of patients with normal kidney function. Our analysis suggests that equations incorporating serum albumin and/or Hb values better estimate the HbA1c-BG relationship in PD patients compared to equations using HbA1c alone.

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Introduction

Hemoglobin A1c (HbA1c) is a clinically important assessment tool for evaluating the association between glycemic control and outcomes in diabetic patients. Two widely used equations for converting HbA1c into mean blood glucose (BG) levels have included the one derived from the Diabetes Control and Complications Trial [DCCT; average glucose (mg/dl) = $35.6 \times \text{HbA1c} - 77.3$] and the A1c-Derived Average Glucose (ADAG) study formula [average glucose (mg/dl) = $28.7 \times \text{HbA1c} - 46.7$], which were defined in populations without underlying kidney disease [1, 2]. The ADAG group concluded that HbA1c is a reliable substitute for average BG and that aside from analytic variation, the only important determinant of the HbA1c is the preceding 3-month average glucose concentration.

The rate of Hb glycation is determined by temperature, serum pH, Hb concentration, BG concentration, and length of exposure to glucose [3]. Given that dialysis patients have significantly altered Hb concentrations and pH levels, the correlation between HbA1c and BG levels in dialysis patients is different than that of patients with normal renal function. Furthermore, shortened erythrocyte life span and accelerated erythropoiesis due to erythropoiesis-stimulating agent use may further alter HbA1c levels in dialysis patients. Indeed, HbA1c was found to underestimate glucose measurements compared to glycated albumin in diabetic patients on dialysis [4–6]. However, HbA1c is still a widely used measure of chronic glycemic control in dialysis patients [3]. Despite the increasing number of diabetic patients on dialysis and the fact that serum albumin levels are lower in patients undergoing peritoneal dialysis (PD) [7], an HbA1c-BG equation formula specific to this population has not yet been derived. Furthermore, the association between HbA1c and BG in PD patients may be different from hemodialysis (HD) patients, given that the former group is exposed to higher dialysate glucose concentrations. In this study, we sought to develop HbA1c-BG equation models specific for PD patients.

Subjects and Methods

Study Population

The data were obtained from DaVita Inc., the second largest dialysis care provider in the United States with approximately 500 outpatient dialysis centers and 40,000 patients across the country. The creation of this national dialysis patient cohort has been described previously [8–13]. A 60-month prevalent cohort (July 2001 through June 2006) of patients undergoing PD patients was studied. Demo-

graphic data and details of medical history were collected, with information on age, sex, race, and presence of diabetes. The study conformed to the Declaration of Helsinki and International Conference on Harmonization of Good Clinical Practice Guidelines.

Patients receiving dialysis for less than 90 days; those without diabetes; with missing values of albumin, glucose, Hb, or HbA1c; and with <3 measurements for each of these laboratory measures were excluded from this analysis. We divided race/ethnicity into four groups: non-Hispanic white, African American, Hispanic, and other. Asian Americans were included in the other race/ethnicity category due to low sample size.

Laboratory Measures

Blood samples were drawn using uniform techniques in all of the dialysis facilities and were transported to a central laboratory in Deland, Florida, typically within 24 h. All laboratory values were measured by automated and standardized methods. Most laboratory values were measured monthly, including serum urea nitrogen, creatinine, albumin, BG, calcium, phosphate, and bicarbonate. HbA1c was measured at least quarterly and provided in National Glycohemoglobin Standardization Program units (%).

Laboratory values in this dataset 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%. All repeated measures of every relevant variable for each patient within each calendar quarter of 13 weeks were averaged to obtain one quarterly mean value for that given variable.

Statistical Methods

Data were summarized using proportions, means ($\pm$ SD) as appropriate. Categorical variables were analyzed with the χ^2 test or Fisher's exact test, and continuous variables were compared using a t test, Mann-Whitney U test, Kruskal-Wallis H test, or ANOVA, as appropriate. Averaged HbA1c values and BG values were compared using regression models with and without adjustment for Hb, serum albumin, patients' age, sex, and/or race/ethnicity. For the creation of the equation, we averaged all the BG including the intervening months' HbA1c levels used in order to calculate individual patients' mean values. In addition to linear models, quadratic, root, log, and exponential regression models were also applied to estimate the relationship between averaged HbA1c and BG values. The bootstrapping method was also used to estimate average BG and corresponding HbA1c for more reliable estimates. The 1,000 resampling procedure with replacement was selected to approximate the sampling distribution of statistics of interest. Adjusted R^2 (R^2_{adj}) 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 sex- and race/ethnicity-based categories to compare the accuracy of the models in each subgroup. The DCCT and the ADAG equations were used as control estimates. All statistical analyses were carried out with SAS, version 9.3 (SAS Institute, Cary, N.C., USA).

Results

Equation Model Creation

We identified 850 PD patients with 4,566 paired measurements of HbA1c and BG over a 5-year period.

Table 1. Baseline patients' characteristics (n = 850)

Characteristics	Overall		Caucasians		African Americans		Hispanics		Other races		p value ^a
	n	%	n	%	n	%	n	%	n	%	
Total	850	100	403	47	178	21	145	17	124	15	
Age, years	58±13		58±12.9		56±11		58±14		59±11		0.11
Sex											
Female	376	44	154	38	102	57	64	44	56	45	0.001
Male	474	56	249	62	76	43	81	56	68	55	
Type of diabetes											
Type 1	233	27	133	33	36	20	29	20	35	28	0.004
Type 2	617	73	270	67	142	80	116	80	89	72	
Dialysis vintage, years	1.9±1.9		1.6±1.3		2.4±2.5		2.1±2.2		2.1±2.0		<0.001
Laboratory findings											
Total protein, mg/dl	6.7±0.6		6.4±0.6		6.9±0.6		6.7±0.6		6.8±0.6		<0.001
Albumin, g/dl	3.5±0.4		3.5±0.4		3.5±0.4		3.5±0.4		3.5±0.4		0.84
Hb, g/dl	12.0±0.9		12.1±0.9		11.8±1.0		12.1±0.9		12.0±0.9		0.002
Glucose, mg/dl	184±52		186±49		178±51		191±58		181±52		0.1
HbA1c, %	7.1±1.2		7.0±1.2		7.1±1.3		7.2±1.2		7.0±1.2		0.32
Dialysis information											
Kt/V	1.39±0.37		1.36±0.34		1.38±0.41		1.44±0.47		1.50±0.30		0.35

^a p value derived from ANOVA and Pearson's χ^2 test.

Baseline characteristics are shown in table 1. The overall cohort mean age was 58 ± 13 years, which was relatively lower in African Americans (56 ± 11 years). The cohort was largely comprised of non-Hispanic whites (47%), African Americans (21%), and Hispanics (17%), with relatively lower contributions from other race/ethnicities such as Asians, Pacific Islanders, and Native Americans. The mean dialysis vintage was 1.9 ± 1.9 years. Seventy-three percent of patients had type 2 diabetes mellitus.

The relationship between the average HbA1c and the average BG is shown in figure 1 and table 2. The scatter plot suggests that there is a linear relationship between HbA1c and BG, similar to the relationship previously reported in nondialysis populations [1, 2]. Next, we compared several equation models with and without adjustment for confounding factors. As shown in table 2, the equation improved when serum albumin was added to the model (R^2_{adj} : 0.446–0.453). The R^2_{adj} further increased after adding Hb, age, and/or race/ethnicity variables, although the degree of improvement was not large. We did not observe an increase in the R^2_{adj} after adding sex as a covariate, nor with employing quadratic, root, log, and exponential mod-

Fig. 1. Comparison between average HbA1c and average BG levels in each patient (n = 850). The R^2_{adj} for the fitted line was 0.446.

els. For convenience of use in the clinical setting, we selected model 3 (equation incrementally adjusted for serum albumin) and model 4 (equation incrementally adjusted for serum albumin and Hb) as the 'simple' models, and model 8 (fully adjusted model) as the 'complex' model.

Table 2. Relationships between the average BG and the average HbA1c

Model	Covariates	Mean ^a		Bootstrapping ^b	
		R ² _{adj}	RMSE	R ² _{adj}	RMSE
1	AG = $-19.8 + 28.8 \times \text{HbA1c}$	0.445	38.359	0.446	38.364
2	AG = $30.7 + 29.1 \times \text{HbA1c} - 4.3 \times \text{Hb}$	0.451	38.166	0.451	38.169
3	AG = $24.1 + 28.6 \times \text{HbA1c} - 12.2 \times \text{Alb}$	0.453	38.102	0.453	38.097
4	AG = $55.3 + 28.8 \times \text{HbA1c} - 10.2 \times \text{Alb} - 3.3 \times \text{Hb}$	0.456	38.005	0.456	38.001
5	AG = $32.6 + 28.5 \times \text{HbA1c} - 12.5 \times \text{Alb} - 0.1 \times \text{age}$	0.453	38.101	0.453	38.095
6	AG = $33.4 + 28.5 \times \text{HbA1c} - 13.4 \times \text{Alb} - 0.1 \times \text{age} + 4.2 \times \text{sex}$	0.454	38.068	0.454	38.061
7	AG = $36.5 + 28.4 \times \text{HbA1c} - 12.4 \times \text{Alb} - 0.1 \times \text{age} + \text{race}$ (-9.1 if AA, -4.6 if others)	0.456	37.991	0.456	37.988
8	AG = $69.5 + 28.7 \times \text{HbA1c} - 10.1 \times \text{Alb} - 3.7 \times \text{Hb} - 0.1 \times \text{age} + \text{race}$ (-10.1 if AA, -5.4 if others)	0.460	37.864	0.460	37.862
9	AG = $72.3 + 28.8 \times \text{HbA1c} - 10.7 \times \text{Alb} - 4.0 \times \text{Hb} - 0.1 \times \text{age} + 4.1 \times \text{sex} + \text{race}$ (-9.4 if AA, -5.2 if others)	0.460	37.834	0.461	37.832
10	AG = $50.0 + 34.5 \times \text{HbA1c} - 0.4 \times (\text{HbA1c})^2 - 10.1 \times \text{Alb} - 3.8 \times \text{Hb} - 0.1 \times \text{age} + \text{race}$ (-10.0 if AA, -5.4 if others)	0.460	37.864	0.460	37.877

AG = Estimated average glucose; Alb: serum albumin; AA = African Americans; others: other races; RMSE = root mean square error. ^a The mean BG and mean HbA1c were used to estimate correlations. ^b The bootstrapping method was used to estimate the average BG and average HbA1c.

BG Estimation

Next, we compared the average BG and estimated BG using our models and previously reported equations (ADAG and DCCT models established for nondialysis populations). Compared to these previously established equations (R^2_{adj} : 0.446 for both ADAG and DCCT models), our simple and complex models showed stronger correlations (R^2_{adj} : 0.454, 0.457, and 0.457 for models 3, 4, and 8, respectively; fig. 2) in PD patients. In addition, the average BG levels estimated by the DCCT and ADAG models were similar to those estimated by our models if patients had serum albumin levels of 4.0 mg/dl and HbA1c levels $\geq 9.0\%$. However, the estimated glucose levels by the previous models were lower if patients had serum albumin levels < 4.0 mg/dl or HbA1c levels $< 9.0\%$ (table 3).

Utility of the Model in Race/Ethnicity-Based Subgroups

Since the number of individuals in each race/ethnicity group was relatively small, it was essential to check the utility of the equation in each race/ethnicity group. As shown in table 4, the R^2_{adj} values between the average

BG and estimated BG using models 3 and 4 in non-Hispanic whites were 0.414 and 0.419, respectively. There were higher correlations in African Americans (R^2_{adj} : 0.474 and 0.473 for models 3 and 4, respectively) and Hispanics (R^2_{adj} : 0.556 and 0.559 for models 3 and 4, respectively) compared to non-Hispanic whites. These data suggested that this model may have particularly greater explanatory power of BG variation in African-American and Hispanic PD populations compared to their non-Hispanic white counterparts.

Discussion

In this relatively large-scale cohort of 850 diabetic PD patients with frequent glucose measurements (4,566 BG samples), we defined new equations of the association between HbA1c and BG, which performed as well as or superior to previously established HbA1c-BG equations. To our knowledge, these are the first HbA1c-BG models specific for PD patients, whereas previous standard formulas were developed for nondialysis patients. Interestingly, the slope of the HbA1c variable in

Fig. 2. Relationship between average BG and estimated BG according to different estimation models. **a** Model 3 (simple): estimated BG (mg/dl) = $24.1 + 28.6 \times \text{HbA1c} - 12.2 \times \text{Alb}$ ($R^2_{\text{adj}} = 0.454$). **b** Model 4 (simple): estimated BG (mg/dl) = $55.3 + 28.8 \times \text{HbA1c} - 10.2 \times \text{Alb} - 3.3 \times \text{Hb}$ ($R^2_{\text{adj}} = 0.457$). **c** Model 8 (com-

plex): BG (mg/dl) = $69.5 + 28.7 \times \text{HbA1c} - 10.1 \times \text{Alb} - 3.7 \times \text{Hb} - 0.1 \times \text{age} + \text{race/ethnicity}$ (-10.1 if African American, -5.4 if not Caucasian, Hispanic, or African American; $R^2_{\text{adj}} = 0.457$). **d** ADAG formula: BG (mg/dl) = $28.7 \times \text{HbA1c} - 46.7$ ($R^2_{\text{adj}} = 0.446$). Alb = Serum albumin.

our model was almost identical to that of the ADAG model. HbA1c has been observed to underestimate glucose measurements in diabetic PD and HD patients compared to glycated albumin [4–6, 14, 15]. Indeed, when we applied the ADAG and DCCT HbA1c-BG equations to our PD cohort, the estimated glucose levels calculated by these previous equations tended to be lower than the levels calculated by our equations (table 3).

Although a number of studies have shown that high and/or low HbA1c levels are associated with poorer outcomes in HD patients [16–20], there has been limited examination of the association between HbA1c and clinical outcomes in PD patients. To our knowledge, only two studies have examined the association between

HbA1c and clinical outcomes in PD patients to date [13, 21]. The issue of glycemic control may be particularly important for PD patients as PD solutions have high glucose concentrations, which may contribute to hyperglycemia in PD patients. Several other metrics of chronic glycemic levels in patients with chronic kidney disease have been identified, such as glycated albumin [22] and 1,5-anhydro-D-glucitol [23]. Nevertheless, HbA1c is still the most widely used glycemic control indicator for diabetic patients with and without kidney disease. A clear understanding of the relationship between BG and HbA1c in dialysis patients is necessary for setting appropriate day-to-day BG goals according to specific HbA1c targets. Our model may be useful for interpreting and using HbA1c as a reliable metric of glycemic control

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

HbA1c %	Alb mg/dl	Estimated average glucose, mg/dl				
		model 3 (+Alb)	model 4 (+Alb, Hb)	model 8 ^a (complex)	DCCT model	ADAG model
5.0	4.0	118	126	127	101	97
5.0	3.5	124	131	133		
5.0	3.0	131	136	138		
6.0	4.0	147	154	156	136	126
6.0	3.5	153	159	161		
6.0	3.0	159	165	167		
7.0	4.0	176	183	185	172	154
7.0	3.5	182	188	190		
7.0	3.0	188	193	195		
8.0	4.0	204	212	213	208	183
8.0	3.5	210	217	219		
8.0	3.0	216	222	224		
9.0	4.0	233	241	242	243	212
9.0	3.5	239	246	247		
9.0	3.0	245	251	253		
10.0	4.0	261	270	271	279	240
10.0	3.5	267	275	276		
10.0	3.0	274	280	281		

All estimated glucose levels were for patients with Hb 10.0 g/dl and 60 years of age. Note that all of these glucose levels are estimated values, not actual measured values. Alb = Serum albumin. ^a In model 8, all estimated glucose levels were for Caucasians (−10.0 if African American, −5.4 if other race).

Table 4. Correlation between estimated glucose and average glucose for patients on PD

Race	n	R ² _{adj} value				
		model 3 (A1c + Alb)	model 4 (A1c, Alb, Hb)	model 8 (complex)	DCCT	ADAG
Caucasians	403	0.414	0.419	0.421	0.406	0.406
African Americans	178	0.474	0.473	0.472	0.468	0.468
Hispanics	145	0.556	0.559	0.561	0.543	0.543
Other races	124	0.421	0.434	0.428	0.422	0.422
Total	850	0.454	0.457	0.457	0.446	0.446

within the PD population despite confounding factors. With regard to equations for HD patients, we have recently reported three models for HD patients: $BG = 59.2 + 29.4 \times \text{HbA1c} - 20.8 \times \text{serum albumin}$ ($R^2 = 0.483$), $BG = 104.8 + 29.7 \times \text{HbA1c} - 18.4 \times \text{serum albumin} - 4.7 \times \text{Hb}$ ($R^2 = 0.486$), and $BG = 82.9 + 30.7 \times \text{HbA1c} - 16.5 \times \text{serum albumin} - 5.4 \times \text{Hb} + 0.3 \times \text{age} + \text{race}$ ($R^2 = 0.491$) [24]. Compared to the equations for PD patients, the coefficient of albumin was larger in HD patients. We could not fully explain why this discrepancy occurs; however, it might be possible that the constant pH environment in PD and continuous exposure of PD solutions with high glucose concentrations may contribute to this discrepancy.

In this study, we proposed three HbA1c-BG equations. We believe model 3 [BG (mg/dl) = $24.1 + 28.6 \times \text{HbA1c} - 12.2 \times \text{serum albumin}$] may be the most straightforward and practical equation for daily use, given that PD patients have fairly uniform Hb levels as recommended by clinical guidelines, and given the significant interaction between anemia, serum albumin, and mortality predictability of HbA1c [13]. In addition, our analysis revealed that careful interpretation of HbA1c is needed in patients with protein-energy wasting and anemia since their HbA1c levels tend to be lower than the levels in dialysis patients [19]. In these patients, equation models which include serum albumin and Hb (model 4 or the complex model) may be useful in estimating their average glucose level. We previously reported that the HbA1c threshold for higher all-cause mortality was lower in Caucasians [13]. One of the reasons for this observation may be due to the differential association between HbA1c and BG across different race/ethnicities, which has been demonstrated in the general population [25]. Indeed, according to our equation model that was inclusive of race/ethnicity, the average BG is higher in Caucasians compared to African Americans with similar HbA1c levels. Although the difference in R^2 between our models and the previous models was not large, compared to the distributions of estimated BG and actual BG in these models, our models showed better associations than the previous models, especially in a usual BG range such as BG of 100–250 mg/dl (fig. 2).

This study has several limitations. First, our data was not based on continuous daily BG monitoring. Although the association between mean BG obtained by continuous glucose monitoring and self-monitoring BG were reported [15], the association between mean BG obtained by continuous glucose monitoring and multiple random BG has not been well established. 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 to the estimation obtained by using time-averaged values, suggesting robustness of our

models. Second, we did not have any data available on medications, PD modality (continuous ambulatory or automated PD), or dialysis prescription. However, the influences of these on our equation models are likely to be comparatively smaller. Third, our HbA1c-BG model might not be valid in certain racial/ethnic subgroups such as Asians due to the low prevalence in our cohort. Fourth, we did not look at the applicability of these equations in nondialysis patients with and without chronic kidney disease. Finally, our population consisted of patients with relatively shorter dialysis vintage. The association between HbA1c and glucose may be different in patients with longer vintage because uremia, nutritional status, and routine use of erythropoietin influence HbA1c levels.

In conclusion, there was a predictable relationship between HbA1c and BG in PD patients. Our new models for PD patients showed stronger correlations between HbA1c and BG compared to previous models. The model inclusive of serum albumin may be better suited for PD patients because of the systematically lower serum albumin levels in this patient population [7]. By providing more reliable estimation of ambient blood glucose levels, our

HbA1c-BG models may allow health care providers and PD patients to better target and achieve optimal glycemic control.

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Disclosure Statement

Dr. Kalantar-Zadeh is the medical director of DaVita Harbor-UCLA/MFI in Long Beach, Calif., USA. Dr. Mehrotra has received grant support and/or honoraria from Baxter Healthcare and DaVita Inc. The other authors have not declared any conflict of interest.

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