Serum 1,5-Anhydroglucitol Concentrations Are a Reliable Index of Glycemic Control in Type 2 Diabetes With Mild or Moderate Renal Dysfunction

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OBJECTIVE—To assess the relationship between 1,5-anhydroglucitol (AG) levels, which are a marker of glycemic control, and stages of chronic kidney disease (CKD).

RESEARCH DESIGN AND METHODS—This was a cross-sectional study with 269 subjects with type 2 diabetes who were divided into four groups based on estimated glomerular filtration rate (eGFR) using Modification of Diet in Renal Disease (eGFRMDRD) formula: 37 in control, 111 in CKD stages 1–2, 78 in stage 3, and 23 in stages 4–5.

RESULTS—The study groups differed significantly with respect to 1,5-AG and lasting plasma glucose (FPG), age, duration of diabetes, blood pressure, HDL, and percentage of antihypertension medication use. Stepwise multivariate regression analyses showed that 1,5-AG levels in the control group, the CKD stages 1–2 group, and the CKD stage 3 group could be explained by HbA1c, age, duration of diabetes, FPG, and antihypertension medication. However, eGFRMDRD was the only independent determinant of 1,5-AG levels in CKD stages 4–5. Logarithmic transformation 1,5-AG values (ln[1,5-AG]) had a significant inverse correlation with HbA1c and FPG levels for CKD stages 1–2 and CKD stage 3 (all P < 0.001). However, associations between ln[1,5-AG] and HbA1c or FPG were insignificant for CKD stages 4–5 (P = 0.274 and P = 0.080, respectively).

CONCLUSIONS—This study demonstrated that 1,5-AG levels do not appear to be influenced by mild or moderate renal dysfunction, suggesting it is a reliable glycemic marker in type 2 diabetes with CKD stages 1–3.

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Strict glucose control in early stages of type 2 diabetes can result in significant clinical benefits, including reduced cardiovascular mortality (1) and improved renal function (2–4). Although HbA1c levels have been the main glycemic marker in the management of type 2 diabetes since the 1980s, other measures of glycemic control are available, such as glycated albumin, fructosamine, and 1,5-anhydroglucitol (1,5-AG). Compared with HbA1c, serum 1,5-AG levels can reflect more recent glycemic control (5) as well as postprandial hyperglycemia (6).

Measuring 1,5-AG levels has several advantages, including retained metabolic inertness, steady-state levels in all tissues, and negligible influence of sampling conditions such as collection time, body weight, age, sex, and food intake of the subjects (7). However, 1,5-AG reabsorption is competitively inhibited in the renal proximal tubule by glucosuria and is excreted in the urine when glucose levels surpass the renal threshold for glucosuria (8), although these studies had small sample sizes. Little information is available on the relationship between 1,5-AG levels and the mild or moderate renal dysfunction commonly found in subjects with type 2 diabetes. Therefore, this study assessed the effects of renal function on the glycemic marker 1,5-AG in type 2 diabetes using classification by chronic kidney disease (CKD) stage.

RESEARCH DESIGN AND METHODS

Subjects and study design

A cross-sectional study was conducted involving individuals with type 2 diabetes who had serum creatinine and spot urine analyses during at least two visits over 3 months and who had 1,5-AG levels available at the Department of Endocrinology and Metabolism of Kangbuk Samsung Hospital in Korea between 2008 and 2010. The estimated glomerular filtration rate (eGFR) was calculated by using the Modification of Diet in Renal Disease (MDRD) formula: eGFRMDRD = 186 × serum creatinine−1.154 × age−0.203 × [0.742 if female]. In accordance with the National Kidney Foundation Disease Outcomes Quality Initiative classification system, CKD was defined on the basis of eGFR and categorized into three groups defined by the cutoff points between stages 2 and 3 as well as between stages 3 and 4. Therefore, stages in the 1–2 CKD group had eGFRs ≥ 60 mL/min/1.73 m²; the stages 3 CKD group had eGFRs < 60 to ≥ 30 mL/min/1.73 m²; and the stages 4–5 CKD group had eGFRs < 30 mL/min/1.73 m² (15). Subjects with an eGFR ≥ 90 mL/min/1.73 m² without proteinuria or history of kidney disease were placed in the control group. Consequently, 269 subjects were grouped as follows: 57 in control, 111 in
**1.5-AG and CKD**

CKD stages 1–2; 78 in stage 3; and 23 in stages 4–5. Subjects with acute renal failure, recent infection, dialysis, known renal tubular acidosis, pregnancy, type 1 diabetes (C-peptide <0.18 nmol/L), chronic liver disease, or malignancy were excluded. Subjects aged <30 or >70 years were also excluded due to the small number of participants in these age categories and potential comorbidities. The protocol for this study was approved by the institutional review board at Kangbuk Samsung Hospital. Written informed consent was obtained from all subjects.

**Laboratory data**

Levels of 1,5-AG were measured using an enzymatic assay with a two-reagent system, Determiner I 1,5-AG (Kyowa Medex, Tokyo, Japan). The assay was automated with an ADVIA 1800 Automatic Analyzer (Siemens, IL) according to the manufacturer’s instructions. The measurement procedures, reference intervals, and coefficients of variation (CV) for 1,5-AG were based on previously published research (16).

HbA1c levels were measured using high-performance liquid chromatography (Variant II, Bio-Rad Laboratories, Hercules, CA). The CV for glucose was 0.05 vs. control, and the three CKD categories using linear regression analyses were performed to identify the independent variables associated with log-transformed 1,5-AG values (ln[1,5-AG]) values. To perform these analyses, demographic variables and continuous variables that were significantly different in baseline characteristics and significantly correlated with ln(1,5-AG) values in the Spearman correlations were included in the model. The model also included dummy variables, such as smoking status, and use of antihypertension and antidysslipidemia medication. Finally, the relationships between ln(1,5-AG) levels, HbA1c, and fasting plasma glucose (FPG) levels were assessed for the control group and the three CKD categories using linear regression analyses. IBM SPSS Statistics 19.0 software (IBM, Armonk, NY) was used for the statistical analyses.

**Table 1—Baseline characteristics of the subjects according to CKD stages**

| Variables                        | Control       | CKD stages 1–2 | CKD stage 3 | CKD stages 4–5 | P       |
|----------------------------------|---------------|----------------|-------------|----------------|---------|
| Demographics                     |               |                |             |                |         |
| Age (years)                      | 54 (47–62)‡§†| 57 (51–62)‡§†| 61 (56–64)*†| 62 (52–68)*    | <0.001  |
| Women                            | 26 (45.6)     | 37 (33.3)      | 36 (46.2)   | 9 (39.1)       | 0.258   |
| Clinical characteristics         |               |                |             |                |         |
| Diabetes duration (years)        | 1.3 (0.8–5.0)∥‡‡| 6.0 (2.0–10.0)*∥‡‡| 10.0 (4.0–15.0)*∥‡‡| 15.0 (10.0–20.0)*∥‡‡| <0.001  |
| BMI (kg/m²)                      | 24.4 (22.7–26.8)| 25.3 (22.8–27.7)| 24.8 (23.2–26.7)| 24.2 (22.5–25.8)| 0.332   |
| Current smoker                   | 18 (31.6)     | 27 (24.8)      | 10 (14.7)   | 3 (23.1)       | 0.165   |
| Laboratory data                  |               |                |             |                |         |
| Serum creatinine (µmol/L)        | 61.9 (53–71)∥‡‡| 80 (71–97)*∥‡‡| 115 (97–133)*∥‡‡| 248 (221–292)*∥‡‡| <0.001  |
| eGFR_{MDRD} (mL/min/1.73 m²)     | 102 (98–109)∥‡‡| 76 (69–88)*∥‡‡| 55 (46–59)*∥‡‡| 22 (14–28)*∥‡‡| <0.001  |
| 1,5-AG (µg/mL)                   | 12.4 (5.5–17.6)∥‡‡| 8.1 (3.0–15.3)*∥‡‡| 1.7 (1.3–9.2)*∥‡| 3.9 (2.0–5.1)*| <0.001  |
| FPG (mmol/L)                     | 7.5 (6.9–9.2)∥‡‡| 8.3 (7.3–10.3)*∥‡‡| 7.7 (6.6–9.1)*∥‡‡| 6.5 (5.3–7.2)*∥‡‡| <0.001  |
| HbA1c (%)                        | 6.8 (6.2–7.9)∥‡‡| 7.4 (6.6–8.7)∥‡‡| 7.4 (6.3–8.0)∥‡‡| 7.8 (6.6–9.5)∥‡‡| 0.071   |
| Systolic BP (mmHg)               | 126 (116–137)*∥‡| 134 (124–143)*∥‡| 132 (120–143)∥‡| 136 (130–150)∥‡| 0.011   |
| Diastolic BP (mmHg)              | 78 (73–89)∥‡‡| 81 (74–91)∥‡‡| 76 (66–81)*∥‡‡| 80 (73–89)*∥‡‡| <0.001  |
| Total                            | 4.8 (4.3–5.3)∥‡‡| 4.7 (4.1–5.4)∥‡‡| 4.5 (3.7–5.3)∥‡‡| 4.3 (3.4–5.3)∥‡| 0.214   |
| HDL                              | 1.2 (1.0–1.4)∥‡‡| 1.2 (1.1–1.4)∥‡‡| 1.1 (0.9–1.3)*∥‡| 1.0 (0.8–1.2)*∥| <0.001  |
| LDL                              | 2.6 (2.1–3.1)∥‡‡| 2.7 (2.2–3.3)∥‡‡| 2.5 (1.8–3.4)*∥‡| 2.4 (1.5–3.0)∥| 0.451   |
| Triglycerides (µmol/L)            | 1.8 (1.2–2.7)∥‡‡| 1.5 (1.1–2.5)∥‡‡| 1.7 (1.2–2.2)∥‡| 1.5 (0.9–2.1)∥| 0.720   |
| Insulin (µmol/L)                  | 64.5 (53.9–75.4)| 66.2 (46.6–99.6)| 64.0 (50.0–82.4)| 47.4 (31.5–93.7)| 0.433   |
| C-peptide (µmol/L)                | 0.88 (0.70–1.02)| 0.89 (0.68–1.10)| 0.84 (0.67–1.10)| 1.07 (0.63–1.62)| 0.757   |
| Presence of albuminuria          | 0 (0)*∥‡‡| 111 (100)*∥‡‡| 37 (47.4)*∥‡| 23 (100)*∥‡| <0.001  |
| Urinary ACR (mg/g)                | 10 (7–15)∥‡‡| 81 (50–174)*∥‡‡| 25 (8–143)*∥‡‡| 376 (200–2,000)*∥‡‡| <0.001  |
| Medications                      |               |                |             |                |         |
| Antihypertension                 | 19 (33.3)     | 67 (60.9)      | 46 (59.7)   | 22 (95.7)      | <0.001  |
| Antidysslipidemia                | 9 (15.8)      | 38 (34.2)      | 35 (45.5)   | 13 (56.5)      | 0.001   |

Values are median with interquartile range or number (proportions). *P value by Kruskal-Wallis test with post hoc analyses or χ² test. ACR, albumin-to-creatinine ratio; BP, blood pressure; *P < 0.05 vs. control; †CKD stage 1; §CKD stages 2–3; and ¶CKD stages 4–5.
used for statistical analyses. Values of $P < 0.05$ were considered statistically significant.

**RESULTS**

**Comparison of baseline clinical variables among groups according to CKD stage**

Baseline demographic and clinical characteristics, laboratory values, and history of medication for participants are reported in Table 1. Duration of diabetes, serum creatinine, eGFRMDRD, and urinary albumin-to-creatinine ratio levels were significantly higher in the CKD groups than in the control, and increased with increasing CKD stage. There were significant differences in 1,5-AG and FPG levels but no significant difference in HbA1c levels among groups.

**Correlations among 1,5-AG, eGFRMDRD, and other clinical variables**

The ln(1,5-AG) values were significantly negatively correlated with FPG and HbA1c levels, except for CKD stages 4–5. However, 1,5-AG was not associated with serum creatinine or eGFRMDRD in a simple correlation analysis (Supplementary Table S1). Analysis of stepwise multivariate regression is reported in Table 2. For the control group and CKD stages 1–3, HbA1c was a consistently explanatory variable of 1,5-AG. FPG, age, duration of diabetes, and medication of antihypertension were significantly associated with 1,5-AG (all $P < 0.001$, irrespective of groups and models). However, eGFRMDRD was the only independent determinant of 1,5-AG levels for CKD stages 4–5.

**Correlations between ln(1,5-AG) and HbA1c or FPG levels according to CKD stages**

Figure 1 illustrates that the inverse correlations between ln(1,5-AG) and HbA1c levels remain significant in the control ($R^2 = 0.534$), CKD stages 1–2 ($R^2 = 0.481$), and CKD stage 3 ($R^2 = -0.483$) groups (all $P < 0.001$), but not for the CKD stages 4–5 group ($R^2 = 0.057$, $P = 0.274$; Fig. 1). Similarly, ln(1,5-AG) is inversely correlated with FPG in the control ($R^2 = 0.414$), CKD stages 1–2 ($R^2 = 0.356$) and CKD stage 3 groups ($R^2 = 0.227$, all $P < 0.001$), but not for the CKD stages 4–5 group ($R^2 = 0.145$, $P = 0.080$; Fig. 2).

**CONCLUSIONS**—Although 1,5-AG appears to be more useful than HbA1c as a short-term marker of glycemic control and more specific in the assessment of postprandial hyperglycemia for subjects with well or moderately controlled diabetes (15,17,18), it has limitations for use in subjects with renal tubular acidosis, uremia, or end-stage renal disease (ESRD). However, the relationship between 1,5-AG levels and mild or moderate renal dysfunction, which is the most common stage of CKD in subjects with type 2 diabetes, is unclear.

The current study detected no relationships between 1,5-AG values and eGFRMDRD for CKD stages 1–3 in a relatively large sample of Korean subjects with type 2 diabetes. Moreover, the associations between 1,5-AG and HbA1c or FPG levels were strongly significant in subjects with CKD stages 1–3, but not in subjects with CKD stages 4–5. This suggests that 1,5-AG levels reflect glycemic state and might be useful for assessing glycemic control if patients are in a state of mild or moderate renal dysfunction.

This study showed that eGFRs have influence on 1,5-AG levels not in subjects with mild or moderate renal dysfunction but in those with severe renal dysfunction or ESRD. Possible contributions of renal tubular damage to serum (or plasma) 1,5-AG levels have been shown in several case–control reports. Some reports have claimed that 1,5-AG levels are not valid for subjects with uremia or chronic renal failure; 1,5-AG levels in these groups were as low as those in a type 2 diabetes group, significantly lower than those in healthy controls (19,20), and negatively correlated with serum creatinine (21). However, the numbers of study participants in these studies were small ($n = 32–79$). On the other hand, a study by Yamanouchi et al. (22) showed that renal dysfunction has little influence on 1,5-AG concentrations. These inconsistent results might be due to group or classification discrepancies. This study was the first to analyze 1,5-AG concentrations in type 2 diabetes classified by CKD stage.

**Table 2**—Stepwise multivariate linear regression analyses as independent determinants of ln(1,5-AG) according to CKD stages

|              | Control | CKD stages 1–2 | CKD stage 3 | CKD stages 4–5 |
|--------------|---------|----------------|-------------|----------------|
|              | $\beta$ | $P$            | $\beta$     | $P$            | $\beta$     | $P$            | $\beta$     | $P$            |
| HbA1c        |         |                |             |                |             | $-0.695$     | $<0.001$     |                |                |
| FPG          |         |                |             |                |             | $-0.702$     | $<0.001$     |                |                |
| Age          |         |                |             |                |             | $0.139$      | $0.049$      |                |                |
| eGFRMDRD     |         |                | $0.720$     | $0.013$        |             |                |                |                |
| HbA1c        |         |                |             |                |             | $0.603$      | $<0.001$     |                |                |
| FPG          |         |                |             |                |             | $0.254$      | $0.008$      |                |                |
| Duration of diabetes |         |                | $-0.233$     | $0.019$        |             |                |                |                |
| eGFRMDRD     |         |                | $0.907$     | $0.012$        |             |                |                |                |

Covariables that are not significant in the models are deleted in the Table. Model 1: age, sex, HbA1c, duration of diabetes, and eGFRMDRD. Model 2: Model 1 plus FPG, systolic blood pressure, HDL, and triglycerides. Model 3: Model 2 plus smoking status and use of antihypertension and antidysslipidemia medication.
Although some have suggested that 1,5-AG levels cannot be used as a reliable index of glycemic control when subjects are in a state of renal dysfunction (14, 20, 23), 1,5-AG levels in this study had significant inverse correlations with HbA1c and FPG levels when subjects had normal to moderate renal dysfunction. However, caution should be taken when using 1,5-AG values as markers of glycemic control in cases of severe renal dysfunction or ESRD. Although little information is available regarding the contribution of renal impairment to the tubular reabsorption of 1,5-AG through sodium glucose cotransporter 4 (SGLT4), a recently described candidate transporter of 1,5-AG (24, 25), it may be conferred from the studies of other glucose transporters. For nephrectomized rats, expression, but not activity, of glucose transporters decreased (26), and glucose transporters in brush-border membranes diminished in spontaneous idiopathic Fanconi syndrome (27). As CKD stage or renal tubular damage progresses, it is possible that resorption of 1,5-AG would be reduced due to a decrease in SGLT4s and aggravated damage of glucose cotransporters. Until now, there were no actual data to support this hypothesis. Further understanding of the characteristics of SGLTs under renal dysfunction is needed.

FPG and 1,5-AG are correlated ($r = 0.381, P = 0.080$), and this relationship is stronger than the correlation between 1,5-AG and HbA1c ($r = -0.238, P = 0.274$) in subjects with CKD stages 4–5. Emoto et al. (14) revealed that inverse correlations between 1,5-AG and HbA1c could be insignificant in the CKD stages 4–5 group, the correlation between 1,5-AG and FPG should explored in future studies with a large sample size.

The main strengths of the present work are the relatively large study sample and the careful characterization of the participants, during which the relationships between 1,5-AG levels and eGFR_mDRD or CKD stages were first evaluated. The limitations of this study are that subjects self-reported their medical history and subjects with renal tubular acidosis were included, which may have confounded the results. In addition, the number of participants with CKD...
stages 4–5 was small. A sophisticated study of a large number of subjects with severe renal impairment or ESRD would help improve the understanding of the effects of renal impairment on 1,5-AG levels.

In conclusion, this study demonstrated that mild or moderate renal dysfunction appears to have no effect on serum 1,5-AG concentrations and that the relationship between 1,5-AG and glycemic markers suggests that 1,5-AG levels may be useful for monitoring glycemic control in subjects with CKD stages 1–3.

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W.J.K. collected and analyzed the data and wrote the manuscript. C.-Y.P. coordinated the study, interpreted the data, contributed to discussion, and reviewed and edited the manuscript. C.-Y.P. 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.

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