Glycemic Control Modifies Difference in Mortality Risk Between Hemodialysis and Peritoneal Dialysis in Incident Dialysis Patients With Diabetes

Results From a Nationwide Prospective Cohort in Korea

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Abstract: Although numerous studies have tried to elucidate the best dialysis modality in end-stage renal disease patients with diabetes, results were inconsistent and varied with the baseline characteristics of patients. Furthermore, none of the previous studies on diabetic dialysis patients accounted for the impact of glycemic control. We explored whether glycemic control had modifying effect on mortality between hemodialysis (HD) and peritoneal dialysis (PD) in incident dialysis patients with diabetes.

A total of 902 diabetic patients who started dialysis between August 2008 and December 2013 were included from a nationwide prospective cohort in Korea. Based on the interaction analysis between hemoglobin A1c (HbA1c) and dialysis modalities for patient survival (P for interaction = 0.004), subjects were stratified into good and poor glycemic control groups (HbA1c < 8.0% or ≥8.0%). Differences in survival rates according to dialysis modalities were ascertained in each glycemic control group after propensity score matching.

During a median follow-up duration of 28 months, the relative risk of death was significantly lower in PD compared with HD in the whole cohort and unmatched patients (whole cohort, hazard ratio [HR] = 0.65, 95% confidence interval [CI] = 0.47–0.90, P = 0.01; patients with available HbA1c [n = 773], HR = 0.64, 95% CI = 0.46–0.91, P = 0.01). In the good glycemic control group, there was a significant survival advantage of PD (HbA1c < 8.0%, HR = 0.59, 95% CI = 0.37–0.94, P = 0.03). However, there was no significant difference in survival rates between PD and HD in the poor glycemic control group (HbA1c ≥8.0%, HR = 1.21, 95% CI = 0.46–2.76, P = 0.80).

This study demonstrated that the degree of glycemic control modified the mortality risk between dialysis modalities, suggesting that glycemic control might partly contribute to better survival of PD in incident dialysis patients with diabetes.

Introduction

Diabetes mellitus is the leading cause of end-stage renal disease (ESRD) worldwide, and the number of diabetic patients who commence dialysis therapy is constantly increasing. Therefore, nephrologists are confronted with the problem of deciding which dialysis modality should be recommended to these patients in clinical practice. Although dialysis modality is usually determined based on a number of factors, including the patient’s clinical condition, socioeconomic status, patient’s or physician’s preference, and the practice pattern of the institution, several other problems, such as vascular access and peritoneal membrane permeability, should be considered before making a decision for ESRD patients with diabetes mellitus. In addition, patient survival rates in hemodialysis (HD) and peritoneal dialysis (PD) are another issue to consider. To date, numerous previous studies have compared differences in the survival rates between diabetic HD and PD patients, but the results were not consistent. Heterogeneity of the study population, diverse dialysis duration, and difference in statistical analysis methods might contribute to these discrepant
findings among previous studies.\textsuperscript{2,5,13,18,19} Recently, in this point of view, the European Renal Best Practice Diabetes Guideline Development Group proposed that there was a lack of evidence in favor or against a particular dialysis modality as the first choice in ESRD patients with diabetes.\textsuperscript{18}

The survival advantage of one dialysis modality over another has been found to vary according to the presence of diabetes.\textsuperscript{4,7,10,12,13} However, none of the previous studies on diabetic dialysis patients accounted for the impact of glycemic control, which is known to be closely associated with clinical outcomes in diabetic patients.\textsuperscript{20–22} In this study, we hypothesized that glycemic control might have an impact on better clinical outcomes of one dialysis modality over another in dialysis patients with diabetes. In contrast to most prior studies in which only baseline demographic characteristics and comorbid diseases were adjusted for,\textsuperscript{4,6,7,14,17} various previously demonstrated independent prognostic factors in dialysis patients, such as high-sensitivity C-reactive protein (hs-CRP), residual renal function (RRF), and hemoglobin A\textsubscript{1c} (HbA\textsubscript{1c}), were included in the final analysis. In result, we attempted to clarify the impact of glycemic control on the association between dialysis modality and patient survival in incident dialysis patients with diabetes.

**METHODS**

**Ethical Statements**

This study was carried out in accordance with the Declaration of Helsinki, and the study protocol was approved by the Institutional Review Board at each participating hospital’s Clinical Trial Centre. All patients provided their written informed consent before entering the study.

**Study Design and Subjects**

All diabetic ESRD patients who started HD or PD between August 1, 2008 and September 30, 2013 at 36 centers of the Clinical Research Center for ESRD (CRC for ESRD) in Korea were initially screened for this study. This study was part of a nationwide multicenter joint network prospective cohort study on ESRD patients in Korea designed to improve survival rates and quality of life and to draw up effective treatment guidelines (clinicaltrial.gov NCT00931970). Patients younger than 18 years, with a history of kidney transplantation before dialysis therapy, with underlying active malignancy or acute infection, or who were expected to survive <3 months were excluded before the initial screening. Among 2035 incident dialysis patients, diabetes mellitus was diagnosed in 1163 patients based on diagnostic criteria of the American Diabetes Association (ADA).\textsuperscript{23} After excluding 261 patients who failed to maintain dialysis for >3 months, 902 patients were included in the final analysis as the whole cohort. To evaluate the impact of glycemic control, 773 patients were included after excluding 129 patients whose mean HbA\textsubscript{1c} levels during the first 6 months were not available (Figure 1).

**Data Collection and Follow-up**

Demographic, clinical and laboratory data were extracted from the electronic data management system. Demographic and clinical data including age, sex, height, weight, comorbidities, smoking history, and body mass index were collected at the time of study entry. The following laboratory data were measured from fasting blood samples at 3 and 6 months after initiation of dialysis, and every 6 months thereafter: white blood cell, hemoglobin, blood urea nitrogen, creatinine, albumin, glucose, etc.

**FIGURE 1.** Flow diagram of patients. Among 1163 incident dialysis patients with diabetes, 902 patients were primarily analyzed as the whole cohort after excluding 261 patients who failed to maintain dialysis for the first 90 days. A total of 773 patients were categorized by HbA\textsubscript{1c} <8.0% or \textgeq 8.0%, then PS matching was performed in each glycemic control group. 1:1 Matching resulted in 199 matched pairs and 36 matched pairs, respectively. HbA\textsubscript{1c} = hemoglobin A\textsubscript{1c}, PS = propensity score.
matching yielded 199 matched pairs in HbA1c 1:1 by PS using a nearest-neighbor matching algorithm. PS All covariates were used for PS matching. Patients were matched pairs in HbA1c rates (the value of HbA1c was used at baseline, 3 months, and 6 months after multivariable logistic regression analysis in each HbA1c group. characteristics according to dialysis modalities. PS was calculated by assigned, we performed propensity score (PS) matching for inter- estis whether glycemic control had a modifying impact on patient survival rates of one dialysis modality over the other, the inter- action of PD and HD in patients with HbA1c <8.0% (log-rank test, \( P = 0.009 \)) (Figure 2A). The 1-, 2-, and 5-year patient survival rates were 92.0%, 84.0%, and 59.0% for HD patients, whereas they were 97.0%, 90.0%, and 65.0% for PD patients, respectively. In 773 patients whose HbA1c concentrations were available, Kaplan–Meier curves also revealed that cumulative survival rates were significantly lower in HD compared with PD patients (log-rank test, \( P = 0.012 \)) (Figure 2B). Cox proportional hazard analysis found that PD was associated with a lower risk of death compared with HD in not only the whole cohort but also the available HbA1c group (whole cohort, HR = 0.65, 95% confidence interval [CI] = 0.47–0.90, \( P = 0.01 \); patients with available HbA1c, HR = 0.64, 95% CI = 0.46–0.91, \( P = 0.01 \)) (Table 2).

Impact of Glycemic Control on Survival Advantage of PD Over HD

PS matching resulted in no differences in baseline characters- teristics between HD and PD in both HbA1c <8.0% (Table 3) and ≥8.0% groups (Table 4). Difference in survival rates between HD and PD varied according to the degree of glycemic control (Figure 3). The cumulative survival rates were significantly lower in HD relative to PD in patients with HbA1c <8.0% (log-rank test, \( P = 0.021 \)) (Figure 3A). However, there was no significant difference in the cumulative survival rates between HD and PD in patients with HbA1c ≥8.0% (log-rank test, \( P = 0.770 \)) (Figure 3B). Furthermore, the survival advantage of PD over HD remained consistent in the good glycemic control group even after PS was adjusted in the multivariate model (HbA1c <8.0%, HR = 0.59, 95% CI = 0.37–0.94, \( P = 0.03 \)) (Table 2). However, patient survival rates were comparable between 2 dialysis modalities in the poor glycemic control group in PS-adjusted model (HbA1c ≥8.0%, HR = 1.21, 95% CI = 0.46–2.76, \( P = 0.80 \)) as well as in the crude model (Table 2).

DISCUSSION

Innumerable studies have demonstrated the survival advantage of one dialysis modality over another in diabetic dialysis patients, but the results have been conflicting. In this study, we demonstrate that PD was significantly associated with decreased risk of all-cause mortality compared with HD in Korean incident dialysis patients with diabetes. Moreover, we found that better patient survival in PD was associated with the
degree of glycemic control. PD patients had a survival advantage in the good glycemic control group, but not in the poor glycemic control group. Even though the choice of dialysis modality is a critical issue, it remains an unsettled question in diabetic dialysis patients. Among a number of factors, patient survival is one important concern to consider before determining the dialysis modality. Several previous studies showed that PD patients had a lower risk of death in the first 1 to 2 years after starting dialysis therapy, which was attributed to preserved RRF in these patients. However, this issue was not fully evaluated because of lack of RRF data in previous studies. In

### TABLE 1. Baseline Characteristics of Patients With and Without HbA1c

|                      | All (n = 902) | Patients With HbA1c (n = 773) | Patients Without HbA1c (n = 129) | P  |
|----------------------|--------------|--------------------------------|---------------------------------|----|
| Age, y               | 62.2 ± 11.9  | 62.2 ± 11.9                    | 61.9 ± 11.9                     | 0.78 |
| Male, n (%)          | 573 (63.5%)  | 492 (63.6%)                    | 81 (62.8%)                      | 0.84 |
| Hemodialysis, n (%)  | 637 (70.6%)  | 538 (69.6%)                    | 99 (76.7%)                      | 0.12 |
| Modified CCI         | 6.2 ± 2.2    | 6.1 ± 2.1                      | 6.4 ± 2.3                       | 0.24 |
| Comorbid disease, n (%) |            |                                |                                 |    |
| CAD                  | 151 (16.7%)  | 130 (16.8%)                    | 21 (16.3%)                      | 0.9 |
| PAD                  | 92 (10.2%)   | 81 (10.5%)                     | 11 (8.5%)                       | 0.64 |
| CVA                  | 95 (10.5%)   | 80 (10.3%)                     | 15 (11.6%)                      | 0.64 |
| CHF                  | 119 (13.2%)  | 109 (14.1%)                    | 10 (7.8%)                       | 0.05 |
| Smoker, n (%)        | 431 (47.8%)  | 379 (49.0%)                    | 52 (40.3%)                      | 0.07 |
| Systolic blood pressure, mmHg | 142.6 ± 22.3 | 142.5 ± 22.5                  | 143.1 ± 21.5                    | 0.80 |
| Diastolic blood pressure, mmHg | 76.4 ± 13.1 | 76.5 ± 13.0                    | 75.9 ± 13.2                     | 0.66 |
| BMI, kg/m²           | 23.4 ± 3.3   | 23.4 ± 3.4                     | 23.5 ± 2.9                      | 0.76 |
| HbA1c (%)            | —            | 6.4 (5.7–7.3)                  | —                               |    |
| White blood cell, ×10^3/μL | 7.4 ± 3.3   | 7.4 ± 3.3                      | 7.1 ± 3.2                       | 0.32 |
| Hemoglobin, g/L      | 90 ± 15      | 90 ± 16                        | 91 ± 15                         | 0.41 |
| Blood urea nitrogen, mmol/L | 27.7 ± 12.5 | 28.2 ± 12.4                    | 25.0 ± 13.2                     | 0.01 |
| Creatinine, μmol/L   | 689.5 ± 291.7 | 689.5 ± 282.9              | 680.7 ± 300.6                   | 0.76 |
| Albumin, g/L         | 32 ± 6       | 32 ± 6                         | 31 ± 5                          | 0.09 |
| Calcium, mmol/L      | 2.0 ± 0.2    | 2.0 ± 0.2                      | 2.0 ± 0.2                       | 0.56 |
| Phosphorous, mmol/L  | 1.7 ± 0.6    | 1.7 ± 0.6                      | 1.7 ± 0.6                       | 0.49 |
| hs-CRP, mg/L         | 0.39 (0.12–1.43) | 0.39 (0.12–1.33)             | 0.72 (0.20–2.48)                | 0.01 |
| ESA use, n (%)       | 615 (68.2%)  | 552 (71.4%)                    | 63 (50.0%)                      | <0.001 |
| RRF, mL/min/1.73 m²  | 8.2 ± 4.4    | 8.2 ± 4.4                      | 8.3 ± 4.7                       | 0.9 |

Data are expressed as mean ± standard deviation, median (interquartile range), or number of patients (%). BMI = body mass index, CAD = coronary artery disease, CCI = Charlson comorbidity index, CHF = congestive heart failure, CVA = cerebrovascular accident, ESA = erythropoiesis-stimulating agent, HbA1c = hemoglobin A1c, hs-CRP = high-sensitivity C-reactive protein, PAD = peripheral artery disease, RRF = residual renal function.

* hs-CRP values were available in 70 patients of patients without HbA1c.

FIGURE 2. Kaplan–Meier analysis of all-cause mortality according to dialysis modality in the (A) whole cohort of 902 patients and (B) 773 patients who had available HbA1c. Patients treated with HD showed significantly higher all-cause mortality than those with PD in the whole cohort (log-rank test, P = 0.009) and in 773 patients (log-rank test, P = 0.012). HbA1c = hemoglobin A1c, HD = hemodialysis, PD = peritoneal dialysis.
our study, RRF was not significantly associated with dialysis modality in terms of patient survival ($P$ for interaction $= 0.24$). Moreover, there were no significant differences in mortality between HD and PD patients in both the lower and higher RRF group (data not shown). Therefore, we surmised that the survival advantage of our PD patients was not linked to RRF. Instead, these findings were more likely attributed to overall improvement of PD outcomes, which was recently observed in many cohort studies.19–26 Mehrotra et al.26 demonstrated that a progressive decline in mortality risk was observed in PD patients between the earlier (1996–1998) and more recent (2002–2004) cohorts. Similarly, a study from the Taiwan showed that diabetic PD patients had worse survival than diabetic HD patients in the 1997 to 2001 cohort. However, the survival difference did not exist in the 2002 to 2006 cohort.14 Although the exact mechanism for this salutary change in PD patient survival is not clear, application of quality-improvement programs in PD, individualization of PD prescription, and reduced risk of PD-related infectious complications have been proposed as reasonable candidates.19

### TABLE 2. HRs of Mortality for Peritoneal Dialysis Compared With Hemodialysis in the Whole Cohort, Unmatched Available HbA$_{1c}$ Group, and PS-matched Group

|                | HR (95% CI) | $P$ |
|----------------|-------------|-----|
| Whole cohort (n = 902) | Crude | 0.65 (0.47–0.90) | 0.01 |
| Unmatched group (n = 773) | Crude | 0.64 (0.46–0.91) | 0.01 |
| PS matched group | | | |
| HbA$_{1c}$ <8.0% (n = 398) | Crude | 0.59 (0.37–0.93) | 0.02 |
| HbA$_{1c}$ ≥8.0% (n = 72) | Adjusted for PS | 1.43 (0.47–2.81) | 0.77 |

CI = confidence interval, HbA$_{1c}$ = hemoglobin A1c, HR = hazard ratio, PS = propensity score.

### TABLE 3. Baseline Characteristics for Unmatched and Propensity Score-Matched Groups in Patients With HbA$_{1c}$ <8.0%

|                | Unmatched | PS-Matched |
|----------------|-----------|------------|
|                | HD (n = 460) | PD (n = 199) | $P$ |
| Age, y         | 64.1 ± 11.7 | 59.0 ± 11.6 | <0.001 |
| Male, n (%)    | 287 (62.4%) | 137 (68.8%) | 0.07 |
| Modified CCI   | 6.2 ± 2.1 | 5.9 ± 2.3 | 0.24 |
| Comorbid disease, n (%) | | | |
| CAD            | 77 (16.7%) | 39 (19.6%) | 0.22 |
| PAD            | 44 (9.6%) | 22 (11.1%) | 0.32 |
| CVA            | 52 (11.3%) | 19 (9.5%) | 0.30 |
| CHF            | 54 (11.7%) | 34 (17.1%) | 0.04 |
| Smoker, n (%)  | 221 (45.9%) | 116 (58.3%) | 0.002 |
| SBP, mmHg      | 144.4 ± 22.9 | 139.7 ± 21.7 | 0.01 |
| DBP, mmHg      | 75.5 ± 13.5 | 78.6 ± 12.8 | 0.01 |
| BMI, kg/m$^2$  | 23.4 ± 3.5 | 23.5 ± 3.3 | 0.81 |
| HbA$_{1c}$ (%) | 6.2 (5.6–6.8) | 6.3 (5.6–6.9) | 0.34 |
| White blood cell, ×10$^3$μL | 7.4 ± 3.7 | 7.3 ± 2.5 | 0.55 |
| Hemoglobin, g/L | 28.8 ± 5.4 | 32.8 ± 5.4 | <0.001 |
| BUN, mmol/L    | 80.7 ± 35.7 | 77.0 ± 34.3 | 0.22 |
| Creatinine, μmol/L | 698 ± 35 | 707 ± 292 | 0.73 |
| Albumin, g/L   | 33 ± 6 | 33 ± 6 | 0.94 |
| Calcium, mmol/L | 2.0 ± 0.2 | 2.0 ± 0.2 | 0.50 |
| Phosphorous, mmol/L | 1.7 ± 0.6 | 1.8 ± 0.6 | 0.51 |
| hs-CRP, mg/L   | 0.39 (0.14–1.50) | 0.39 (0.11–1.24) | 0.04 |
| ESA use, n (%) | 348 (75.7%) | 131 (65.8%) | 0.007 |
| RRF, mL/min/1.73m$^2$ | 8.0 ± 4.3 | 8.1 ± 3.9 | 0.79 |

Data are expressed as mean ± standard deviation, median (interquartile range), or number of patients (%). BMI = body mass index, BUN = blood urea nitrogen, CAD = coronary artery disease, CCI = Charlson comorbidity index, CHF = congestive heart failure, CVA = cerebrovascular accident, DBP = diastolic blood pressure, ESA = erythropoiesis-stimulating agent, HbA$_{1c}$ = hemoglobin A1c, HD = hemodialysis, hs-CRP = high-sensitivity C-reactive protein, PAD = peripheral artery disease, PD = peritoneal dialysis, PS = propensity score, RRF = residual renal function, SBP = systolic blood pressure.
| Unmatched PS-Matched | Unmatched | PD | PD | P |
|----------------------|-----------|----|----|---|
| **Age, y**           | 61.8 ± 11.9 | 57.2 ± 11.3 | 0.04 | 58.5 ± 12.1 | 57.2 ± 11.3 | 0.56 |
| **Male, n (%)**      | 47 (60.3%) | 21 (58.3%) | 0.84 | 23 (63.9%) | 21 (58.3%) | 0.63 |
| **Modified CCI**     | 6.4 ± 2.2 | 6.3 ± 1.7 | 0.42 | 6.4 ± 2.5 | 6.3 ± 1.7 | 0.72 |
| **Comorbid disease, n (%)** |    |    |    |    |    |    |
| CAD                  | 10 (12.8%) | 4 (11.1%) | 0.79 | 3 (8.3%) | 4 (11.1%) | 0.69 |
| PAD                  | 11 (14.1%) | 4 (11.1%) | 0.66 | 5 (13.9%) | 4 (11.1%) | 0.72 |
| CVA                  | 6 (7.7%) | 3 (8.3%) | 0.91 | 4 (11.1%) | 3 (8.3%) | 0.69 |
| CHF                  | 14 (17.9%) | 7 (19.4%) | 0.85 | 5 (13.9%) | 7 (19.4%) | 0.53 |
| Smoker, n (%)        | 37 (47.4%) | 15 (41.7%) | 0.69 | 18 (50.0%) | 15 (41.7%) | 0.48 |
| SBP, mmHg            | 142.4 ± 21.8 | 134.9 ± 20.1 | 0.15 | 135.6 ± 18.5 | 134.9 ± 20.1 | 0.95 |
| DBP, mmHg            | 77.1 ± 11.2 | 76.1 ± 10.6 | 0.47 | 77.1 ± 11.6 | 76.1 ± 10.6 | 0.68 |
| BMI, kg/m²           | 23.0 ± 3.3 | 23.2 ± 3.6 | 0.93 | 23.3 ± 3.5 | 23.2 ± 3.6 | 0.78 |
| HbA1c (%)            | 9.0 (8.4–9.8) | 9.1 (8.2–9.9) | 0.79 | 9.0 (8.3–9.8) | 9.1 (8.2–9.9) | 0.72 |
| White blood cell, ×10⁹/µL | 7.9 ± 2.9 | 7.6 ± 2.5 | 0.74 | 7.5 ± 2.6 | 7.6 ± 2.5 | 0.67 |
| Hemoglobin, g/L      | 94 ± 14 | 97 ± 19 | 0.42 | 97 ± 15 | 97 ± 19 | 0.97 |
| BUN, mmol/L          | 26.7 ± 10.9 | 27.1 ± 10.5 | 0.84 | 27.2 ± 11.7 | 27.1 ± 10.5 | 0.81 |
| Creatinine, μmol/L   | 610 ± 221 | 566 ± 159 | 0.34 | 575 ± 194 | 566 ± 159 | 0.82 |
| Albumin, g/L         | 31 ± 6 | 31 ± 6 | 0.67 | 32 ± 6 | 31 ± 6 | 0.89 |
| Calcium, mmol/L      | 2.0 ± 0.2 | 2.0 ± 0.2 | 0.65 | 2.0 ± 0.2 | 2.0 ± 0.2 | 0.45 |
| Phosphorus, mmol/L   | 1.7 ± 0.5 | 1.7 ± 0.5 | 0.25 | 1.7 ± 0.4 | 1.7 ± 0.5 | 0.72 |
| hs-CRP, mg/L         | 0.39 (0.10–0.68) | 0.24 (0.05–0.65) | 0.29 | 0.39 (0.15–0.70) | 0.24 (0.05–0.65) | 0.20 |
| ESA use, n (%)       | 53 (67.9%) | 20 (55.6%) | 0.14 | 19 (52.8%) | 20 (55.6%) | 0.81 |
| RRF, mL/min/1.73m²   | 8.9 ± 3.8 | 10.3 ± 7.7 | 0.23 | 9.2 ± 4.8 | 10.3 ± 7.7 | 0.31 |

Data are expressed as mean ± standard deviation, median (interquartile range), or number of patients (%). BMI = body mass index, BUN = blood urea nitrogen, CAD = coronary artery disease, CCI = Charlson comorbidity index, CHF = congestive heart failure, CVA = cerebrovascular accident, DBP = diastolic blood pressure, ESA = erythropoiesis-stimulating agent, HbA1c = hemoglobin A1c, HD = hemodialysis, hs-CRP = high-sensitivity C-reactive protein, PAD = peripheral artery disease, PD = peritoneal dialysis, PS = propensity score, RRF = residual renal function, SBP = systolic blood pressure.
It is noteworthy that the survival advantage of PD was altered by the degree of glycemic control in diabetic patients starting dialysis therapy. The survival advantage of one dialysis modality over the other varied according to the presence of diabetes. However, so far, no study has explored the impact of glycemic control, which is closely related with clinical outcomes in diabetic patients, on the difference in patient survival between the two dialysis modalities in diabetic dialysis patients. In our study, the better survival of PD patients was observed only in patients with HbA1c <8.0%, suggesting that the survival benefit of PD was robust in the good glycemic control group. The mechanism by which glycemic control exerts an impact on the mortality of PD relative to HD in diabetic ESRD patients can somewhat be explained by peritoneal damage from hyperglycemia and adherence to treatment. Since PD patients are exposed to a large amount of glucose absorbed from the dialysate, continuous exposure to dialysate might worsen glycemic control and induce peritoneal damage in diabetic patients. Indeed, high peritoneal membrane transport characteristic along with increased protein permeability is more commonly accompanied in diabetic PD patients than nondiabetic PD patients. Although an objective assessment of peritoneal damage according to the degree of glycemic control was not performed, we inferred that the detrimental effect of poor glycemic control may accelerate peritoneal damage and late diabetic complications. This, in turn, lessened the survival benefit of PD in patients with HbA1c ≥8.0%. Meanwhile, since PD is a home-based modality, a patient’s adherence to treatment and their ability to follow instructions are crucial to maintaining therapy. Adherence to therapy was revealed to be associated with the risk of hospitalization and mortality in dialysis patients; in other words, compliant patients had a lower mortality risk. Moreover, a recent study showed that patients starting PD had increased risks of hospitalization and peritonitis, especially in the early period. These findings suggest that adaptation of self-care education is important to reduce mortality risk. Taken together, we suggest that patients in the good glycemic control groups were more compliant, and thus were able to achieve self-care, which may partly contribute to the survival advantage of PD.

The present study has several limitations. First, even though the PS matching method was used to overcome limitations of nonrandom allocation to dialysis modality, residual confounding effect cannot be totally excluded. It was difficult to examine whether the indication bias of dialysis modality selection exerted any influence on clinical outcomes. However, to date, most studies included only demographics and comorbidities. In contrast, various dialysis-related prognostic factors, including biochemical variables such as hemoglobin, albumin, calcium, phosphorous, and hs-CRP, as well as RRF were used in this study for PS matching to mitigate confounding effect. Second, as HbA1c values at baseline, 3 months, and 6 months after dialysis initiation were not available in 129 patients, these patients were excluded from the final analysis of PS matching. But there were no significant differences between patients with and without mean HbA1c levels except the use of erythropoiesis-stimulating agent (ESA). Third, shortened erythrocyte survival and ESA use can discredit HbA1c as a marker of glycemic control in dialysis patients. To overcome this limitation, hemoglobin concentrations and ESA use were included in PS calculation and it was revealed that there were no significant differences in hemoglobin levels and ESA use between HD and PD after PS matching. Recent evidence indicated that HbA1c had modest-to-strong correlations with serum glucose, glycated albumin, and serum fructosamine, and these correlations were similar between HD and PD patients. Glycated albumin and fructosamine have been known to be impervious to anemia than HbA1c, but unfortunately, these surrogates were not available in our cohort. Fourth, the HbA1c cutoff value of 8.0% used to stratify patients into good and poor glycemic control groups was relatively arbitrary. However, as our subjects were incident dialysis patients and the mean value of HbA1c at dialysis initiation and during the first 6 months was used, HbA1c <8.0% was defined as the good glycemic control group according to the ADA guideline for patients with advanced micro or macrovascular complications (stage 3 or worse chronic kidney disease). Fifth, as this study included only Korean incident dialysis patients, results may not be generalized to other populations. Primary outcome in the present study was relatively small compared with those in Western

FIGURE 3. Kaplan–Meier analysis of all-cause mortality according to dialysis modality in the (A) PS-matched good glycemic control group (HbA1c <8.0%) and (B) PS-matched poor glycemic control group (HbA1c ≥8.0%). Cumulative survival rates were significantly lower in patients on HD compared with PD in good glycemic control group (n = 398, log-rank test, P = 0.021). However, there was no significant difference in survival rates between HD and PD in poor glycemic control group (n = 72, log-rank test, P = 0.770). HbA1c = hemoglobin A1c, HD = hemodialysis, PD = peritoneal dialysis, PS = propensity score.
ESRD patients. We surmised that the difference may be attributed to disparate ethnicities, as the mortality rates of our patients were comparable with those of Japanese patients. Lastly, the follow-up duration was relatively short. Notwithstanding these limitations, the present study has distinct strengths. The heterogeneity of the study population has been indicated as a possible reason for discrepant findings among previous studies. According to a recent systematic review of 25 eligible studies by the European Renal Best Practice Diabetes Guideline Development Group, no study included only diabetic patients, the proportion of diabetic patients ranged from 9% to 61%. On the contrary, we included only incident dialysis patients with diabetes to focus the impact of glycemic control in these patients and to preserve sufficient statistical power. Finally, the present study enrolled nationally distributed subjects from private clinics to tertiary hospitals between 2008 and 2013, suggesting that the results of this study on our nationwide contemporary cohort could be more helpful to provide evidence for deciding optimal dialysis modality for Korean diabetic dialysis patients in the present era.

In conclusion, the overall patient survival of PD was significantly higher compared with that of HD in incident dialysis patients with diabetes. In addition, better patient survival with PD was influenced by the degree of glycemic control. Survival advantage of PD patients was consistent only in the good glycemic control group. These findings suggest that glycemic control may partly contribute to better survival of PD in Korean incident dialysis patients with diabetes. Nevertheless, further well-designed randomized controlled studies are required to delineate the causal relationship between glycemic control and dialysis modality-related mortality in these patients.

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