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Peritoneal dialysis assists residual renal function to maintain glucose tolerance: a prospective observational study

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Abstract

Background: Renal failure can cause the deterioration of glucose metabolism and increase blood glucose fluctuations, which have been reported to be a factor in the progression of vascular disorders. Little information is available on the daily variation in blood glucose levels in patients with end-stage renal disease (ESRD) and peritoneal dialysis (PD).

Methods: Daily plasma glucose excursion was evaluated by continuous glucose monitoring (CGM) for 24–72 h. Fourteen ESRD patients without type 2 diabetic nephropathy (non-diabetic patients) and six ESRD patients with type 2 diabetic nephropathy (diabetic patients) participated in this study. ESRD patients started PD which were undergone in only the daytime, and not in the night time. As the PD solution, Dianeal®1.5 % PD-2N (1.5 L) was infused intraperitoneally for 3 h three times daily. The patients received standard meals with a total calorie intake of 30 kcal/kg/day and a protein intake of 0.8 g/kg/day before and after initiation of PD. Daily glucose profiles were measured both 2 week before and 1 week after the commencement of PD with CGM system (CGMS).

Results: CGMS parameters were compared between before and after the initiation of PD. In non-diabetic patients with ESRD, the maximum glucose concentration (GC) and area under the curve (AUC) of GC >140 mg/dL significantly decreased after PD initiation. In diabetic patients with ESRD, the mean GC, SD, minimum value, AUC of GC >140 mg/dL, and maximum nocturnal GC significantly decreased, while the percentage of nocturnal GC <70 mg/dL markedly increased after PD commencement. In all patients, there was a significant negative correlation between the incidence of hypoglycemia and residual renal function before the initiation of PD and the correlation disappeared after PD initiation. In all patients, the GC range and coefficient of variation were negatively associated with the peritoneal Kt/V value.

Conclusions: Residual renal function promotes the maintenance of glucose tolerance in patients with ESRD. Glucose fluctuation improved shortly after initiation of PD, whereas nocturnal hypoglycemia was evident in diabetic patients.

Keywords: Blood glucose fluctuation, End-stage renal disease, Peritoneal dialysis, Residual renal function
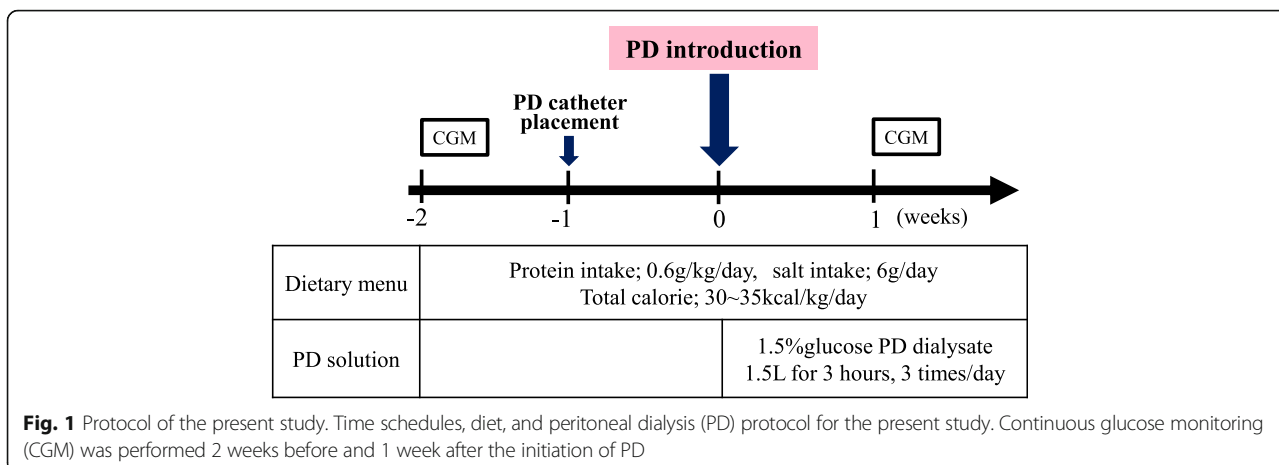
Background

In patients with renal failure, glucose metabolism is abrogated, and fluctuation in plasma glucose levels is significantly enhanced because of increased insulin resistance [1–7]. Recent clinical studies have demonstrated that acute glucose excursions, postprandial hyperglycemia, and hypoglycemia, in addition to mean blood glucose levels, have a major influence on vascular endothelial function and are related to cardiovascular disease through the promotion of oxidative stress [8–11]. In in vitro research, apoptosis of human umbilical vein endothelial cells was shown to increase during glucose fluctuations as compared to during constant high glucose concentrations (GCs) [12]. However, blood glucose fluctuations of patients with end-stage renal disease (ESRD) have not been precisely evaluated [13, 14].

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Fig. 1 Protocol of the present study. Time schedules, diet, and peritoneal dialysis (PD) protocol for the present study. Continuous glucose monitoring (CGM) was performed 2 weeks before and 1 week after the initiation of PD

Table 1 Baseline characteristics and biochemical parameters before and after PD

Variables	ESRD (n = 20)			
	Non-diabetic (n = 14)		Diabetic (n = 6)	
Age (years)	58.07 ± 12.85		58.17 ± 7.17	
Sex (male/female)	11/3		6/0	
BMI (kg/m^2)	23.54 ± 2.90		29.76 ± 6.42	
Antihypertensive agents				
ARB/ACE inhibitor	6 (42.86 %)		6 (100 %)	
Ca channel blocker	12 (85.71 %)		4 (66.67 %)	
Hypoglycemic drugs				
Insulin			4 (66.67 %)	
Dpp-4 inhibitor			1 (16.67 %)	
Sulfonylurea			0 (0 %)	
α-GI			2 (33.33 %)	
Biochemical parameters	Before PD	After PD	Before PD	After PD
Total protein (mg/dL)	6.02 ± 0.98	6.39 ± 0.71*	5.60 ± 0.86	6.17 ± 0.50*
Albumin (g/dL)	3.38 ± 0.75	3.43 ± 0.59	2.93 ± 0.62	3.15 ± 0.56
BUN (mg/dL)	71.64 ± 16.70	53.08 ± 11.64**	67.47 ± 20.76	48.10 ± 12.62**
Creatinine (mg/dL)	10.90 ± 3.93	9.96 ± 3.46*	10.50 ± 1.85	10.08 ± 2.21
e-GFR (mL/min/1.73 m^2)	4.89 ± 1.91		4.84 ± 1.39	
HbA1c (%)	5.33 ± 0.33	5.58 ± 0.39	6.22 ± 1.32	6.43 ± 1.25
GA (%)	15.60 ± 4.18	16.90 ± 3.41	15.84 ± 2.78	16.20 ± 3.05
WBC (10^3/μL)	6.38 ± 1.65	6.01 ± 1.14	7.85 ± 1.84	7.43 ± 1.45
Hb (g/dL)	9.80 ± 1.08	10.40 ± 0.87	10.12 ± 0.87	11.08 ± 0.70*
CRP (mg/dL)	0.14 ± 0.13	0.37 ± 0.63	0.52 ± 0.90	0.35 ± 0.46

Results are shown as mean ± SD

ESRD end-stage renal disease, *BMI* body mass index, *ACE* angiotensin-converting enzyme, *ARB* angiotensin-receptor blocker, *Dpp-4 inhibitor* dipeptidyl peptidase 4 inhibitor, *α-GI* α-glucosidase inhibitor, *BUN* blood urea nitrogen, *e-GFR* estimated glomerular filtration rate, *HbA1c* hemoglobin A1c, *GA* glycoalbumin, *WBC* white blood cell, *Hb* hemoglobin, *CRP*, C-reactive protein

**$p < 0.01$ vs. before PD; *$p < 0.05$ vs. before PD

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The recently developed continuous glucose monitoring system (CGMS) allows precise assessment of daily blood glucose fluctuations, hypoglycemia, and hyperglycemia [15–20]. The present study was performed to determine the blood glucose profiles of patients with ESRD before and after the initiation of peritoneal dialysis (PD) in order to evaluate the effects of this dialysis modality. Furthermore, residual renal function plays a key role in regulating the blood glucose level of patients with ESRD [5, 6, 21, 22]. Focusing on these points, the blood glucose level was measured before and at 1 week after introduction of PD in the prospective study. This is the first study to compare glucose metabolism in patients with ESRD before and after the initiation of PD, which causes dynamic changes in systemic glucose homeostasis.

Methods

Aims

The aims are to reveal the exacerbation of blood glucose fluctuation in ESRD patients and how peritoneal dialysis affects the glucose metabolism in the induction period.

Participants

Twenty patients without infectious diseases including an exit-site infection and peritonitis, with a mean age of 59.63 years, participated in this study. Six patients had type 2 diabetic nephropathy and 14 patients had no history of diabetes, in our medical record. They underwent CGM after we acquired their informed consent.

Study design and protocol

This study was conducted as a prospective observational study. The CGM device used in this study was an iPro®
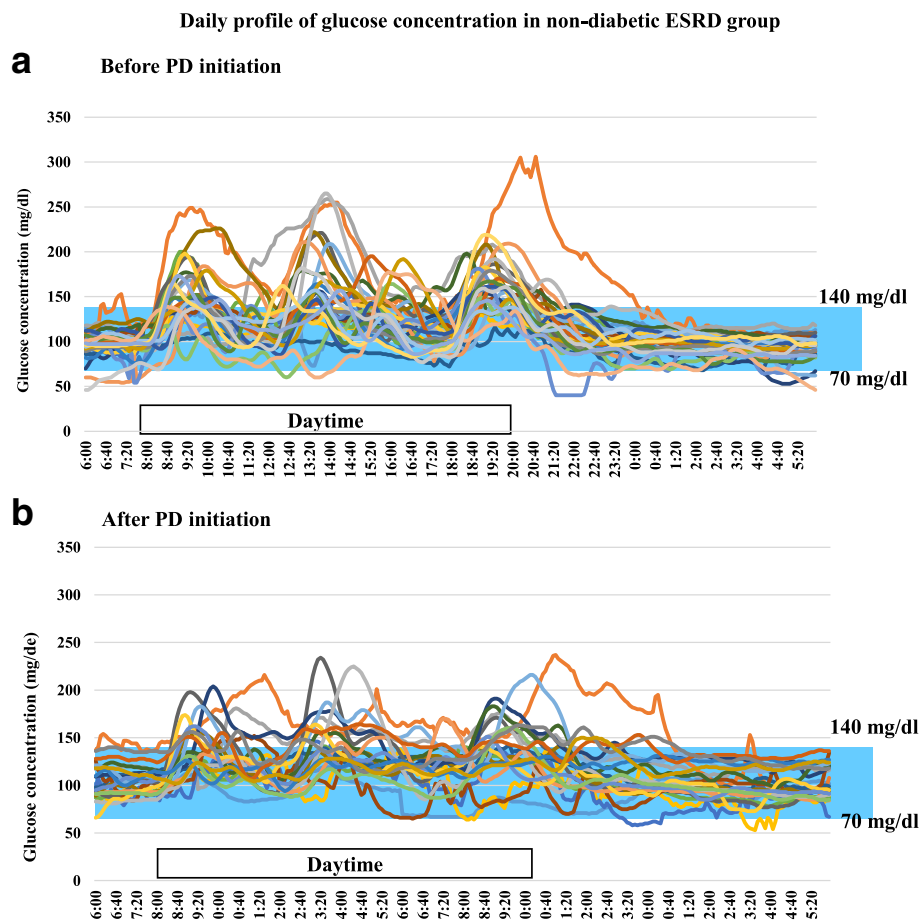
Fig. 2 Daily glucose concentration (GC) profile based on continuous glucose monitoring (CGM) in non-diabetic end-stage renal disease (ESRD) patients. **a** Daily CG profile for each ESRD patient without diabetes before peritoneal dialysis (PD) initiation (*n* = 14). **b** Daily CG profile for each ESRD patient without diabetes after PD initiation (*n* = 14). The *x*-axis represents the time at which GC was measured. The *y*-axis represents GC level. Each *line* corresponds to the result of one subject. The GC range of 70–140 mg/dL is shadowed in *blue* in the graph. The daytime period, which ranges from 0800 h to 2000 h, is indicated by a box on the *x*-axis

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2 CGM (Medtronic, Minneapolis, MN). The recorder of the iPro® 2 is small ($3.5 \times 2.8 \times 0.9$ cm), lightweight (5.7 g), and waterproof, allowing it to be comfortably worn on the body. All CGM data are strongly correlated with blood glucose levels because they are adjusted for data obtained every 8 h by self-monitoring of blood glucose levels [23–25]. The protocol of the study is shown in Fig. 1. Baseline CGM data were obtained 2 weeks before the initiation of PD by continuous monitoring for 24 to 72 h. CGM was repeated 1 week after the initiation of PD and was performed for 24 to 72 h. During CGM, PD was performed by infusing 1.5 L of 1.5 % glucose dialysate solution (Dianeal, Baxter, Chicago, IL) for 3 h, three times per day, starting at 10:00 AM. A PD catheter was inserted 1 week before the initiation of dialysis. None of the subjects developed any infections, including catheter exit-site infections, while CGM was performed, before and after the initiation of PD. None of the subjects were positive for C-reactive protein at the time of CGM. All subjects were hospital patients receiving a standard hospital diet, regardless of PD status, with a protein intake of 0.8 g/kg, salt intake of 6 g/day, and total calorie intake of 30 kcal/kg/day. No snacks were allowed between meals. This diet was provided throughout the present study. We also confirmed the participants ate almost all the diet, in our medical record. Because retention of dialysate for PD induces abdominal distension, food consumption might be reduced. In the present study, dialysate was only retained in the peritoneal cavity for 9 h a day, so distension-associated reduction of food consumption did not occur. Anti-diabetic therapy, including oral hypoglycemic reagents or insulin injection, was not changed after PD initiation. This study was approved by the Ethics Committee of Keio University School of Medicine (the IRB number; 20120065) and was registered with the University Hospital Medical Information Network (UMIN) clinical trial registry before the start of the study (ID: UMIN000013483). After each patient was provided with a detailed explanation of the study, informed consent to participate was obtained.

Statistics

Statistical analyses were performed using Microsoft Office Excel 2007 and IBM SPSS statistics 22.0 software (IBM Corp., Armonk, NY). The Kolmogorov-Smirnov test was used to examine the normality of data distribution, after which a paired t test, ANOVA, or a nonparametric procedure (Wilcoxon signed-rank test) was used to confirm statistically significant differences, with $p < 0.05$ indicating significance. Results are expressed as the mean ± standard deviation (SD). The mean blood glucose levels of each patient were calculated and compared before and after the introduction of PD.

Therefore, the effects of differences in the duration of CGM measurement before and after introduction of PD were excluded.

Results

Baseline characteristics of the subjects and biochemical parameters before and after PD in all patients

Baseline characteristics of the patients without diabetic nephropathy with ESRD (non-diabetic ESRD, $n = 14$) and patients with type 2 diabetic nephropathy with ESRD (diabetic ESRD, $n = 6$) are shown in Table 1. Among the two groups, mean age, sex ratio, and body mass index were similar. With respect to oral antihypertensive medications, non-diabetic ESRD patients were primarily taking calcium channel blockers, and all diabetic ESRD patients were taking ARBs. Diabetic ESRD patients were treated primarily with insulin or α-glucosidase inhibitors as hypoglycemic drugs.

In non-diabetic ESRD patients, TP increased significantly after the introduction of PD, while BUN and creatinine decreased significantly. In diabetic ESRD patients, TP and Hgb increased significantly after initiation of PD, while BUN declined significantly after its introduction (Table 1).

Table 2 The CGMS parameters of non-diabetic patients with ESRD

CGMS parameters	ESRD (non-diabetic) ($n = 14$)	
	Before PD	After PD
Mean GC (mg/dL)	120.81 ± 19.29	116.30 ± 18.07
SD of GC (mg/dL)	26.22 ± 13.14	21.63 ± 7.17
Range of GC (mg/dL)	117.57 ± 57.82	95.07 ± 35.92
Minimum GC (mg/dL)	75.79 ± 20.80	89.86 ± 44.10
Maximum GC (mg/dL)	193.36 ± 55.64	162.50 ± 42.72*
AUC of GC >140 mg/dL (mg · min/dL)	6.98 ± 11.04	4.16 ± 6.96*
Percentage of GC <70 mg/dL	4.70 ± 7.09	5.51 ± 8.02
Percentage of GC ≥70 to <180 mg/dL	88.74 ± 11.37	91.31 ± 9.07
Percentage of GC ≥180 mg/dL	6.56 ± 11.74	3.19 ± 7.08
Mean nocturnal GC (mg/dL)	107.53 ± 16.64	106.64 ± 22.73
SD of nocturnal GC (mg/dL)	12.96 ± 9.60	13.58 ± 9.10
Range of nocturnal GC (mg/dL)	57.36 ± 40.58	53.14 ± 29.01
Minimum nocturnal GC (mg/dL)	84.36 ± 20.12	85.64 ± 15.93
Maximum nocturnal GC (mg/dL)	141.71 ± 37.56	138.79 ± 36.58
Percentage of nocturnal GC <70 mg/dL	6.68 ± 13.91	10.19 ± 20.48
Percentage of nocturnal GC ≥70 to <180 mg/dL	91.22 ± 14.96	86.18 ± 22.76
Percentage of nocturnal GC ≥180 mg/dL	2.10 ± 7.79	3.64 ± 13.35

Results are shown as mean ± SD
AUC area under the curve, *CGMS* continuous glucose monitoring system, *ESRD* end-stage renal disease, *GC* glucose concentration, *PD* peritoneal dialysis, *SD* standard deviation
*$p < 0.05$ vs. before PD

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CGM results before and after initiation of PD in non-diabetic ESRD patients

The daily GC profiles before and after initiation of PD in non-diabetic patients are shown in Fig. 2a, b, respectively. The data for each CGM parameter before and after the initiation of PD are listed in Table 2. The maximum GC level and the area under the curve (AUC) of GC >140 mg/dL significantly decreased after the initiation of PD. Other CGM parameters were not altered after initiation of PD. These data indicated that the increased glucose excursion in patients with ESRD was partially ameliorated by PD.

CGM results before and after initiation of PD in diabetic ESRD patients

The profiles of GC changes in diabetic ESRD patients before and after PD initiation are shown in Fig. 3a, b, respectively. Data for each CGM parameter before and after PD initiation are listed in Table 3. The mean GC level, SD, AUC of GC >140 mg/dL, and maximal

nocturnal GC levels significantly decreased after the initiation of PD. Of note, in diabetic ESRD patients, minimum GC levels significantly decreased, and the percentage of nocturnal GC <70 mg/dL significantly increased after PD initiation. In diabetic patients, PD resolved glucose fluctuations, similar to what was observed in non-diabetic ESRD patients. However, nocturnal GC levels tended to be lower after the initiation of PD, which was attributed to nocturnal hypoglycemia.

Correlations of biochemical and PD-related parameters with CGM parameters

The correlations between biochemical and CGM parameters were examined in non-diabetic ESRD patients and diabetic ESRD patients. The correlation between CGM parameters and the index of dialysis efficiency, including peritoneal Kt/V, renal Kt/V, and total Kt/V, were also analyzed. In all patients, the incidence of hypoglycemia (GC <70 mg/dL) was negatively correlated with residual renal function before the start of PD (Fig. 4a,
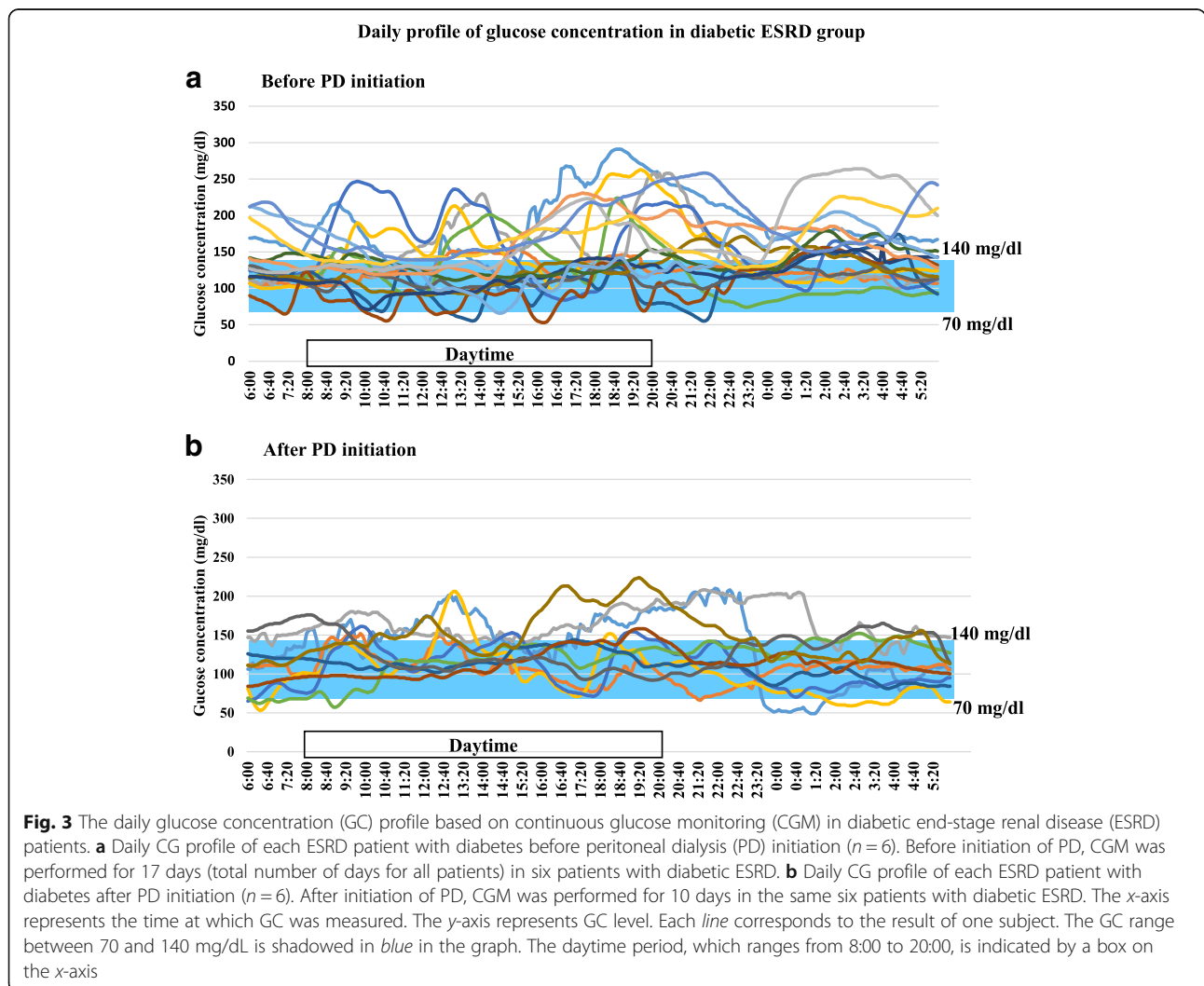
Fig. 3 The daily glucose concentration (GC) profile based on continuous glucose monitoring (CGM) in diabetic end-stage renal disease (ESRD) patients. **a** Daily CG profile of each ESRD patient with diabetes before peritoneal dialysis (PD) initiation (*n* = 6). Before initiation of PD, CGM was performed for 17 days (total number of days for all patients) in six patients with diabetic ESRD. **b** Daily CG profile of each ESRD patient with diabetes after PD initiation (*n* = 6). After initiation of PD, CGM was performed for 10 days in the same six patients with diabetic ESRD. The *x*-axis represents the time at which GC was measured. The *y*-axis represents GC level. Each *line* corresponds to the result of one subject. The GC range between 70 and 140 mg/dL is shadowed in *blue* in the graph. The daytime period, which ranges from 8:00 to 20:00, is indicated by a box on the *x*-axis

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Table 3 CGMS parameters of diabetic patients with ESRD

CGMS parameters	Before PD (n = 6)	After PD (n = 6)
Mean GC (mg/dL)	148.26 ± 25.47	128.18 ± 23.33*
SD of GC (mg/dL)	35.63 ± 13.23	26.84 ± 9.65*
Range of GC (mg/dL)	153.33 ± 55.80	116.33 ± 38.17
Minimum GC (mg/dL)	81.83 ± 20.37	77.50 ± 28.27*
Maximum GC (mg/dL)	235.17 ± 54.16	193.83 ± 30.75
AUC of GC >140 mg/dL (mg · min/dL)	21.29 ± 18.24	9.49 ± 10.24**
Percentage of GC <70 mg/dL	1.44 ± 2.64	8.41 ± 8.69
Percentage of GC ≥70 to <180 mg/dL	77.54 ± 17.20	81.46 ± 11.35
Percentage of GC ≥180 mg/dL	21.03 ± 18.42	10.14 ± 12.10
Mean nocturnal GC (mg/dL)	146.95 ± 30.66	120.89 ± 34.62
SD of nocturnal GC (mg/dL)	26.40 ± 12.94	27.49 ± 18.55
Range of nocturnal GC (mg/dL)	100.83 ± 49.47	86.17 ± 38.39
Minimum nocturnal GC (mg/dL)	108.67 ± 36.72	80.50 ± 29.74
Maximum nocturnal GC (mg/dL)	209.50 ± 49.14	166.67 ± 37.54*
Percentage of nocturnal GC <70 mg/dL	1.24 ± 2.11	21.99 ± 19.52*
Percentage of nocturnal GC ≥70 to <180 mg/dL	77.80 ± 24.88	61.11 ± 30.60
Percentage of nocturnal GC ≥180 mg/dL	20.97 ± 25.74	16.90 ± 28.74

Results are shown as mean ± SD

AUC area under the curve, *CGMS* continuous glucose monitoring system, *ESRD* end-stage renal disease, *GC* glucose concentration, *PD* peritoneal dialysis, *SD* standard deviation

**p < 0.01 vs. before PD, *p < 0.05 vs. before PD

left; $R = -0.539$, $p = 0.014$). However, there was no significant correlation between the incidence of hypoglycemia and residual renal function after commencing PD (Fig. 4a, right). In all patients, after the initiation of PD, peritoneal Kt/V showed a significant correlation with the GC coefficient of variation as well as with GC range (Fig. 4b), indicating that peritoneal function has some influence on GC fluctuation, regardless of diabetic status. After the introduction of PD, the blood glucose level and the SD were 130.89 ± 19.67 mg/dL and 17.81 ± 8.50, respectively, when PD solution was retained for 9 h (between 10:00 and 19:00). Before the introduction of PD, the blood glucose level and the SD was 137.45 ± 27.83 mg/dL and 22.54 ± 12.86, respectively (between 10:00 and 19:00). There were no significant differences of blood glucose and SD between before and after the introduction of PD (130.89 ± 19.67 vs 137.45 ± 27.83, $p = 0.247$ and 17.81 ± 8.50 vs 22.54 ± 12.86, $p = 0.062$, respectively).

Discussion

The GC data obtained by CGM in the present study revealed that ESRD patients had abnormal glucose metabolism, even when diabetes was not present. In these patients, both mean GC and the GC SD increased. Progression of renal failure causes a deterioration of insulin sensitivity [3–7], and improvement can occur

after hemodialysis (HD) or PD [6, 26, 27]. Dialysis also increases the insulin sensitivity of pancreatic β cells and peripheral tissues [6], which can contribute to restoration of β cell function [21, 28, 29]. Several underlying mechanisms have been suggested to explain uremia-associated defects in insulin sensitivity. Urea induces reactive oxygen species that exacerbate insulin resistance in mouse visceral adipose tissue [7]. Other uremic factors, including acidemia, have also been reported to influence glucose metabolism [22, 30, 31]. In the present study, due to these uremic factors, daily GC levels and daily GC fluctuations in the CGMS were increased in non-diabetic patients.

It is conceivable that PD itself is disadvantageous for the control of systemic blood glucose levels, since the PD solution contains 1.5 % glucose (1500 mg/dL). However, in this study, it was revealed that there was little deterioration in glucose metabolism while PD was performed. In the early phase after introduction of PD, there were no significant differences of the mean blood glucose levels and SD at the times with retention of PD solution (from 10:00 to 17:00) between before and after introduction of PD. It has been reported that, unlike with PD solutions containing 4.25 % glucose, PD solutions containing 1.5 % glucose did not cause excessive β cell stimulation and had few adverse effects on glucose metabolism [32]. Although the peritoneum is highly permeable and absorption of glucose occurs rapidly, free water and uremic substances that can induce insulin resistance can be rapidly and simultaneously removed, thereby preventing an increase in blood glucose. DeFronzo et al. have reported that HD improves insulin resistance [6], while it has also been reported that uremic toxins, p-cresyl sulfate and urea, induce insulin resistance [3, 7]. The balance between the addition of glucose dialysate and the removal of free water and uremic substances may prevent the elevation of GC, despite the glucose loading from the PD solution.

Our data also suggested that hypoglycemia occurs in diabetic patients because insulin resistance is increased due to reduced renal function, leading to hyperinsulinemia, while hypoglycemia may occur in non-diabetic patients due to reduction of renal gluconeogenesis related to renal dysfunction [33–35]. This suggests that residual renal function can have an important influence on the blood glucose profile. The incidence of hypoglycemia was lower among patients who had better residual renal function before initiation of PD. After the start of PD, such an influence was no longer observed. Because PD reduced uremic toxicity, the tendency for patients with impaired renal function to be prone to develop hypoglycemia completely disappeared (Fig. 4a, right). The effect of renal dysfunction may be attributable to retention of insulin or uremic toxins that impair glucose

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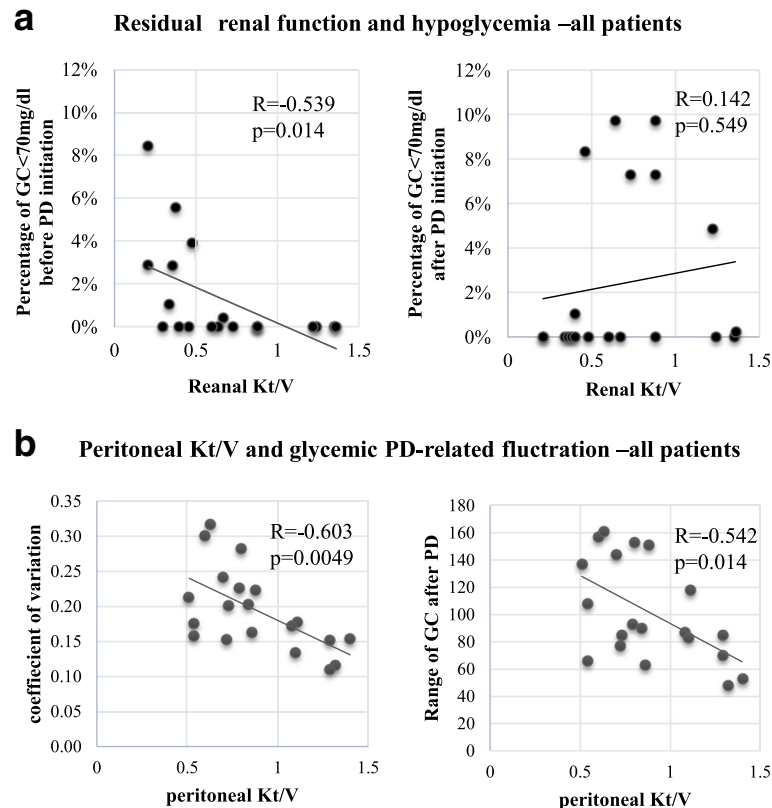
Fig. 4 The relationships between biochemical and peritoneal dialysis (PD)-related parameters and continuous glucose monitoring (CGM) parameters. **a** Correlation between the percentage of GC <70 mg/dL and renal Kt/V, before and after the initiation of PD. **b** Correlation between the PD-related parameters and CGM parameters in all ESRD patients

tolerance, and PD may compensate for the effect. During long-term PD, residual renal function might decline further and PD might not be able to remove sufficient uremic toxins, which might then induce insulin resistance and inflammation. Consequently, glucose in PD dialysates might increase the blood glucose level. But further research is needed to verify this assumption. The peritoneal Kt/V value was negatively correlated with the parameters for GC excursion, the coefficient of variation, and range of GC. Although glucose load increases with high peritoneal efficiency, uremic substances might have been more efficiently removed. This escape from the uremic condition is more important for GC fluctuations. These findings suggest that PD may reduce the risk of hypoglycemia over the medium to long term after initiation, not shortly after initiation, especially in patients with low residual renal function.

HD has also been shown to improve both β cell sensitivity to glucose and tissue sensitivity to insulin [6]. However, HD is an intermittent treatment that is performed for several hours at a time, three times per week. Therefore, HD causes large fluctuations in the levels of uremic toxins and body fluid homeostasis,

and it is not well understood how such fluctuations affect blood glucose levels [26, 27] and nocturnal hypoglycemia [36]. On the other hand, PD is performed slowly and continuously, leading to more stable uremia correction as compared to that with HD. Unlike the PD protocol in the present study, if dialysate is retained in the peritoneal cavity overnight as when ordinary PD is performed, PD might also be able to reduce the occurrence of nocturnal hypoglycemia. Hypoglycemia in patients with chronic kidney disease or patients on dialysis could increase the risk of stroke [37]. The ACCORD study suggested that severe hypoglycemia was likely to result in cardiovascular events [38]. Moreover, a meta-analysis revealed that severe hypoglycemia was associated with a 2.05-fold higher risk of cardiovascular disease, while a bias analysis of the data showed that severe hypoglycemia was correlated with an increased risk of cardiovascular events, unless the underlying disease was related to both severe hypoglycemia and cardiovascular risk factors [39]. A large-scale study showed that ESRD patients on PD had a better prognosis than did those on HD for several years after the initiation of dialysis therapy [2, 40, 41]. This

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difference may be partly attributed to the advantageous effects of PD on glucose metabolism. On the other hand, our study highlights the risk of hypoglycemic adverse effects in diabetic ESRD patients at the initiation of PD. The incidence of nocturnal hypoglycemia was significantly higher after the initiation of PD in patients with ESRD due to diabetic nephropathy. In patients with diabetic ESRD, HOMA-IR was high before the initiation of PD and declined after PD was commenced, although the decrease was not significant (data not shown). In patients with non-diabetic ESRD, HOMA-IR was not particularly high before the initiation of PD and showed little change after PD was commenced. These findings appear to suggest that the incidence of nocturnal hypoglycemia might be higher in patients with diabetic ESRD, after the initiation of PD. Physicians should be aware of the risk of nocturnal hypoglycemia, especially in diabetic patients. In the present study, the mean HbA1c was 4.82 ± 0.37 in patients with hypoglycemia and 5.66 ± 0.81 in patients without hypoglycemia, and the difference was significant ($p = 0.039$). Therefore, we suggest that the target HbA1c was 5.6 %. However, further research will be needed to confirm this conclusion.

Limitations of this study are it is a single-center study and a small-sized study. However, this study was significant because this is the first study evaluating the blood glucose fluctuation before and after the PD initiation under the same condition in meals and PD menu.

Conclusions

We found that daily GC fluctuation was disturbed in ESRD patients irrespective of their diabetic condition and that PD had a beneficial effect on blood glucose levels. We also demonstrated that the risk of nocturnal hypoglycemia was increased, particularly in diabetic patients at the initiation of PD. To detect these changes in glucose metabolism, monitoring GC with CGMS is recommended in ESRD patients, especially when they begin peritoneal dialysis therapy.

Abbreviations

AUC: Area under the curve; CGM: Continuous glucose monitoring system; Non-diabetic patients: ESRD patients without type 2 diabetic nephropathy; Diabetic patients: ESRD patients with type 2 diabetic nephropathy; GC: Glucose concentration; HD: Hemodialysis; PD: Peritoneal dialysis; SD: Standard deviation; UMIN: University Hospital Medical Information Network

Availability of data and materials

Authors' contributions

NW and TK equally contributed to this study. NW, TK, and SW designed this study protocol. NW, TK, SW, and HI analyzed all the data and derived the conclusions. NW and SW drafted the manuscript. KH and HT participated in the data acquisition and helped lead to the conclusions by their advices. All authors read and approved the final manuscript.

Competing interests

NW belongs to the donated fund laboratory, Department of Integrated Renal Replacement Therapy in School of Medicine, Keio University, which was established by a fund from Baxter Co., Ltd. The other authors declare that they have no competing interests.

Consent for publication

Informed consent to publish was obtained from all individual participants included in this study.

Ethics approval and consent to participate

All procedures performed in this study were in accordance with the ethical standards of the Ethics Committee of Keio University School of Medicine at which this study was conducted (IRB approval number 20120065) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Written informed consent to participate was obtained from all individual participants included in this study.

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