Glucose absorption from peritoneal dialysate is associated with a gain in fat mass and a reduction in lean body mass in prevalent peritoneal dialysis patients

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Abstract

The majority of peritoneal dialysates use glucose to generate an osmotic gradient for the convective removal of water and sodium. Although glucose can potentially be absorbed, previous studies have failed to establish whether this leads to increased fat weight gain. We measured body composition using bioimpedance in peritoneal dialysis (PD) patients, electively starting PD, attending for their first assessment of peritoneal membrane function after 2-3 months, and then after 12 months. We studied 143 patients; 89 (62.2%) males, 53 (37.1%) diabetics, mean age 61.3±14.9 years, with 90 (62.1%) of patients treated by automated PD cyclers (APD) with a daytime icodextrin exchange and 37 (25.9%) by continuous ambulatory PD (CAPD). Median fat mass increased by 1.8 (-0.5 to 4.1) kg, whereas fat free mass fell -1.3 (-2.9 to 1.0) kg, and the increase in fat mass was negatively associated with the fall in soft lean mass (r-0.41, $p<0.001$). Increased fat mass was associated with measured peritoneal glucose absorption ($r=0.69$, $p<0.001$), and glucose absorption was associated with the amount of 22.7 g/L glucose dialysate (odds ratio (OR) 2.0, 95% confidence limits (CL) 1.5-2.5, $p<0.001$), peritoneal urea clearance (OR 9.5 (CL 2.4-37.1) $p=0.001$), and male gender (OR 4.8 (CL 1.5-14.9) $p=0.008$). We report an observational study in prevalent PD patients following body composition from their first assessment of peritoneal dialysis membrane function for approximately 12 months, and despite the majority of patients prescribed icodextrin, we have demonstrated an association between intra-peritoneal glucose absorption and fat weight gain, but also loss of fat-free mass.

Introduction

World-wide almost 300,000 patients with end-stage kidney disease are treated by peritoneal dialysis (PD). Clearance of the waste products of metabolism that accumulate in PD patients with end-stage kidney disease is achieved by transport from capillaries in the peritoneal cavity to the peritoneal dialysate, with small solutes moving predominantly by diffusion, and larger solutes and fluid by convection. The majority of PD dialysates use glucose to provide the osmotic driving force for the convective removal of water and sodium. However, higher glucose concentrations in the peritoneal dialysate can lead to glucose diffusing from the peritoneal cavity back into the peritoneal capillaries. The amount of glucose potentially absorbed will potentially depend upon the glucose concentration of the dialysates, cycle dwell time, fill volume and peritoneal transporter status [1]. PD patients are advised to consume 35 kcal/kg/day, although it is accepted that glucose absorption from the dialysate may provide 400-800 kcal/day, and this should be considered as part dietary intake [2,3]

There is debate as to whether the potential glucose load from the peritoneal dialysate leads to changes in body composition. Multiple observational studies from both Northern and Southern Europe, South America and Asia have all reported no association between PD dialysate glucose exposure and increased body fat [4-9], and similarly a prospective Dutch study comparing patients initiating dialysis showed no difference in weight gain between those starting haemodialysis compared to PD [10]. However, studies comparing the impact of using one icodextrin exchange have reported a greater increase in weight and body fat in patients treated by continuous ambulatory peritoneal dialysis (CAPD) prescribed only glucose dialysates compared to the icodextrin group [11,12].

Many of these reports came from small studies in CAPD patients which estimated glucose absorption based on the number of peritoneal glucose exchanges. As such we wished to determine whether measured glucose absorption was associated with changes in body composition measured with bioimpedance [13], in a population treated by both CAPD and automated peritoneal cyclers (APD), after their first assessment of peritoneal membrane function, with a PD prescription designed for their peritoneal transporter status [14].

Patients and methods

We audited the results obtained when adult PD patients who had electively started PD attended for their first routine outpatient assessment of peritoneal membrane function [14], 2-3 months after catheter insertion and then when they returned 11-13 months later for a subsequent assessment of peritoneal membrane function. Prior to starting PD all patients had attended a specialised clinic for patients with chronic kidney disease stage 4 and 5. Patients who had a delayed 1st or 2nd peritoneal membrane assessment due to PD peritonitis or an acute illness requiring hospitalisation, and those prescribed chemotherapy were excluded (Figure 1).

Peritoneal transport assessment used a standard 2 litre 22.7 g/L dextrose exchange Baxter (Baxter Health Care, Deerfield, USA) and body composition by bio-impedance was measured after drainage of peritoneal dialysate. No patient had suffered peritonitis in the three months prior to assessment. Patients with amputations, cardiac pacemakers or defibrillators were excluded from study as bio-impedance measurements were not made. Multi-frequency bioelectrical impedance (MF-BIA) analysis used an 8 tactile electrode system, placed on both hands and both feet [15,16]. Height was measured by a standard wall mounted measure (Sigmeas 1, Edward DohertyBeverley, UK). Peritoneal dialysis bags were weighed pre-infusion, post infusion and upon drainage using calibrated scales (MPSS250, Marsden, Henley on Thames, UK).

Corresponding samples of spent dialysate effluent and serum biochemistry samples were analysed with a standard multi-channel biochemical analyser, including glucose, creatinine using an enzymatic method to adjust for the potentially high glucose levels, and albumin using the bromocresol green method (Roche Integra, Roche diagnostics, Lewes, UK), [17,18]. Twenty-four-hour urine collections were analysed to determine urine volume and residual renal function, and to determine weekly dialysis dose calculated as weekly Kt/Vurea and litres of creatinine cleared/1.73m^2, and normalised protein nitrogen appearance (nPNA) was calculated by standard methods [14,19]. The volume of PD dialysate used was

determined by the in-flow volume recorded by APD cyclers, and for CAPD patients were instructed to allow 15 seconds for the flush before fill, and we measured this volume in the sitting position, with a median volume of 90 mL, which was used to adjust for the volume of dialysate, and glucose instilled for CAPD patients. Glucose absorption was calculated by deducting the glucose in drained out in the 24-hour peritoneal dialysate effluent from the total instilled. Patient PD prescriptions were altered according to modality and patient transport characteristics of the first assessment of peritoneal membrane function, but then maintained until the follow-up bioimpedance measurements. Glucose absorption was calculated at the time of the repeat assessment of body composition.

We used the Stoke-Davies co-morbidity and Rockwood frailty scales [20,21].

Ethics

Our retrospective audit complied with the UK National Health Service (NHS) guidelines for clinical audit and service development with all patient data anonymised and complied with UK National Institute for Clinical Excellence (NICE) best practices: www.nice.org.uk/media/796/23/bestpracticeclinicalaudit.pdf.

Statistical methods

Statistical analysis used the D'Agostino Pearson test for analysis of normality, followed by Chi square adjusted for small numbers and Anova or Kruskal Wallis, with appropriate correction for multiple testing, by Tukey or Games Howell methodology. Univariate analysis was by Spearman's correlation. A logistic regression model on absorption above and below the median, based on variables associated with glucose absorption of $p \leq 0.1$ was constricted, with variables then excluded if not significant in a step-backward, analysis unless they improved the model. strength (SPSS version 22.0 IBM, Armonk, New York, USA and GraphPad Prism version 8.1, San Diego, USA). Data are presented as mean ± standard deviation, median (inter quartile range), or mean and 95% confidence limits (CL), or as a percentage.

Statistical power calculation

For our exploratory observational study of peritoneal dialysis patients in a single centre we reviewed the number of patients reported in earlier studies, which ranged from 20-85 [2-6, 10]. The number of peritoneal dialysis patients in a single centre in the United Kingdom is far less than for haemodialysis. We aimed to include twice as many patients as the earlier studies and were able to report on 143 patients who had electively started on peritoneal dialysis.

Results

We reviewed the results from 206 patients starting PD between April 2005 and December 2016. Sixty-three patients were excluded due to missing glucose absorption or bioimpedance data (Figure 1). We analysed the data from 143 PD patients; 89 (62.2%) males, 54 (37.8%) females, 53 (37.1%) diabetics, with an average age of 61 years. The majority of patients; 90 (62.1%) were treated by automated PD cyclers (APD) with a daytime icodextrin exchange and 37 (25.9%) by continuous ambulatory PD (CAPD) (Table 1). No patient was prescribed 38.6 g/L or 42.5 g/L glucose dialysates. Overall there was an increase in fat mass and a fall in fat free mass after a follow-up of 11-13 months from the first to the second assessment of peritoneal membrane function.

Dividing patients above and below the median glucose absorption, then patients with greater glucose absorption were more likely to be male and treated by APD, have less residual renal function and with greater use of 22.7 g/L glucose dialysates, and peritoneal ultrafiltration (Table 2). Those patients with greater glucose absorption had a greater increase in fat weight and loss of fat free mass.

We then compared PD modality, and both patients treated by CAPD and those by APD with a day-time exchange were faster peritoneal transporters and prescribed more 2.27 g/L dialysates and icodextrin compared to those treated by overnight APD, and the APD patients had greater renal urea clearance (Kt/V_{urea}) (Table 2). Fat mass did not differ, but

patients treated by APD with a day time exchange had greater fat free mass. Fat mass generally increased with all PD modalities whereas fat free mass fell.

Spearman univariate correlation coefficients demonstrated a significant association between glucose absorption and the prescription of 22.7 g/L PD dialysates, number of PD cycles, peritoneal urea clearance, body surface area, and fat free mass, and negative association with residual renal function, nPNA and blood sugar (Table 3). Increasing peritoneal glucose absorption was associated with an increase in percentage body fat ($r=0.214$, $p<0.05$) and reduction in lean body mass index ($r=-0.231$, $p<0.01$). Glucose absorption was greater for males 209.4 (98.9-292.7) mmol/day compared to females 98.3 (26.3-241.6) $p=0.004$, and the change in fat mass was greater for male patients ($r=0.32$, $p=0.002$) (Figure 2). Similarly, the loss of fat free mass was greater for males (Figure 3), as was soft lean mass ($r=-0.29$, $p=0.006$), compared to females ($r = -0.10$, $p=0.46$). However, for both genders there was an association between a gain in fat weight and loss of fat free mass (Figure 4). There was no association between glucose absorption, change in fat mass or lean body mass and either co-morbidity or frailty scores. Adjusting fat mass for height, then there was an association between glucose absorption and the change in fat mass index ($r=0.17$, $p=0.04$). Comparing patients above and below the median glucose absorption, then those with higher glucose absorption had a greater increase in fat mass index ($X2=6.9$, $p=0.032$).

A multivariable regression model comparing patients above and below the median glucose absorption showed that higher glucose absorption was associated with increased prescription of 22.7 g/L glucose PD dialysates, greater peritoneal urea clearance, male gender, and for non-diabetics (table 4). Similarly, a greater increase in fat mass index was associated with 22.7 g/L glucose dialysates (β 0.08, standard error β 0.03, standardised β 0.19, 95% confidence limits 0.008 to 0.14)) and negatively with nPNA (β -1.35, standard error β 0.61, standardised β -0.19, 95% confidence limits -2.56 to -0.14)).

Discussion

PD patients aged < 60 years are advised to consume 3500 kcal/day, and those > 60years 3000 kcal/day, but this includes glucose absorbed from the dialysates. In clinical practice there are now both glucose containing and non-glucose containing dialysates, so although the median amount of glucose absorbed in our cohort was 124 kcal/day, this ranged from a net loss of glucose up to 534 kcal/day. Many previous studies from Europe, Asia and South America have all failed to demonstrate an effect of glucose administration on increasing body fat [4-10]. One of the confounders is that patients may be uraemic prior to starting PD and malnourished, so gain weight as a consequence of the reduction in uraemic toxins and improved nutrition [9]. In addition, assessment of peritoneal membrane function is often delayed several weeks after initiation of PD [1,14], so PD prescriptions may not initially be the most appropriate for the invidual patient. To reduce these confounding effects, we studied patients who had been previously reviewed in a specialist clinic preparing patients for dialysis prior to an elective initiation of PD, and then waited until after their first assessment of peritoneal membrane function, so that PD prescriptions were altered for transporter status [1,14], and then after approximately 12 months, when patients attended for further assessment of peritoneal membrane function. Similarly, to avoid the potential confounding effects of acute illness on body composition we excluded patients with PD peritonitis and those with acute hospital admissions.

Previous estimates have proposed that CAPD patients using a combination of 13.6 g/L and 22.7 g/L could potentially absorb around 238 mmol glucose/day and patients using an APD cycler with all glucose exchanges, including a day-time glucose exchange 488 mmol/day [22]. As we used icodextrin exchanges, and also took into account the flush-before fill technique then the amount of glucose exposure was reduced in our CAPD patients compared to earlier studies which used all glucose exchanges and did not account for the flush-before fill technique [22,23]. Rather than following many previous studies which simply used the amount of glucose in the dialysate prescribed [7,9,24], we calculated glucose absorption, by measuring the 24-hour peritoneal glucose balance and found that patients with

greater peritoneal glucose absorption gained more fat weight and lost more fat-free mass over time. Previous studies which have used bioimpedance to assess changes in body composition, have reported no increase in fat weight associated with glucose exposure [7,9]. However, these studies included incident patients and bioimpedance measurements made with peritoneal dialysate instilled within the abdomen, which is reported to lead to inaccuracy in assessments of body composition, with an over estimation of fat weight [25,26].

We report the largest cohort of PD patients starting PD electively and demonstrate that not only did increasing intra-peritoneal glucose absorption lead to an increase in body fat, but also a reduction in the measurements of fat free mass and soft lean mass. This was greatest for patients with less residual renal function who required greater peritoneal urea clearance and ultrafiltration, and larger sized patients, males more than females. Larger sized patients, typically males, and those with less residual renal function were prescribed more 22.7 g/L glucose dialysates.

On the other hand, patients with diabetes were less likely to have an increase in fat weight, as presumably higher blood glucose concentrations reduced the peritoneal to blood gradient and potentially reduced glucose absorption. Previous studies have commented on diabetic PD patients being at increased risk of increases in extracellular water, but not fat weight gain [27,28].

Glucose absorption and the prescription of 22.7 g/L glucose dialysates were greater for those patients treated with APD and a day time icodextrin exchange, and although the median increase in fat weight was higher and loss of fat free mass greater for these patients it was not statistically different from that of patients treated by APD and CAPD. Similarly, peritoneal transport status did not differ between patients treated by CAPD and those by APD with a day time icodextrin exchange, and peritoneal transporter status was not a significant factor for peritoneal glucose absorption. After the first assessment of peritoneal membrane function PD prescriptions were altered according to peritoneal transporter status [1,13], such as shortening dwell times for faster transporters, and as such could have reduced the potential for greater glucose absorption.

Many previous reports failed to demonstrate an association between peritoneal glucose exposure and a gain in body fat [8,29,30]. However, studies replacing a single 2.27 g/L PD exchange with icodextrin have demonstrated a reduction in fat weight [11,12], supporting a role for intraperitoneal glucose absorption and fat weight gain.

Whereas several studies which recruited incident patients reported an increase in both muscle mass and fat gain, or similar increases compared to incident haemodialysis patients [31,10], in our prevalent cohort we noted that fat weight gain was associated with a reduction in muscle mass. Previous reports have suggested that PD patients have low levels of active energy expenditure [32,33], and whether those who gain fat are less active requires further exploration.

Our male patients absorbed around an average of 37.6 g/day (209 mmol/day) of glucose, which would equate to 150 kcal/day and potentially more than 50,000 kcal in a year. Assuming an equivalent for fat gain (9500 kcal/kg), then if patients did not change their diet or physical activity levels, then this could have led to a 5 kg increase i in fat mass. However, the average fat mass gain was 2 kg, and previous studies have reported that PD patients have a blunted hunger profile compard to haemodialysis patients and healthy controls [34], which would potentially suggest that glucose absorption from the dialysate reduces enteral calorie intake. We were unable to measure the physical activity of our patients and did not have an accurate assessment of dietary intake. However, we found no association between glucose absorption or changes in body composition compared to frailty, using the Rockwood frailty score which is based on physical capability [21], and similarly no association with co-morbidity [20]. On the other hand, we noted that there was a negative association between peritoneal glucose absorption and nitrogen appearance rate, suggesting that patients reduced dietary protein intake rather than increased energy expenditure. In addition, models used to calculate changes in fat mass often do not take into account the effect of starting body composition [35], and have not been validated for our dialysis population.

We report an observational study in a cohort of patients electively started on PD with pre-dialysis care in a specialised chronic kidney disease clinic. By following these patients from after their first assessment of peritoneal dialysis membrane function for approximately 12 months, and despite the high proportion of patients prescribed icodextrin and avoidance of the very hypertonic glucose PD dialysates, we have demonstrated an association between intra-peritoneal glucose absorption and fat weight gain, and also a loss of fat-free weight and other estimates of muscle mass. Increased peritoneal glucose absorption was associated with a reduction in nitrogen appearance rate, suggesting a reduced dietary protein intake. Our observational study suggests a hypothesis which will require prospective testing.

The authors have no conflict of interest

Author contributions
AD conceived the audit
AD applied for audit registration and approvals
SL collated and analysed primary data
AD and SL analysed secondary data
SL and AD contributed to and approved final manuscript draft

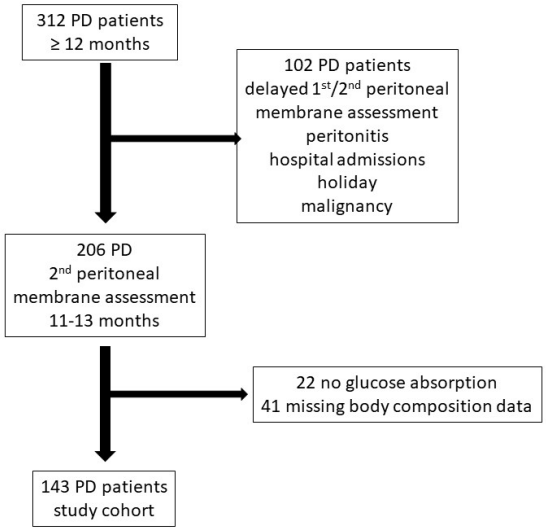

Figure 1. Consort diagram showing study patient recruitment, starting with all patients who electively started peritoneal dialysis (PD) were treated by PD for a minimum of 12 months.

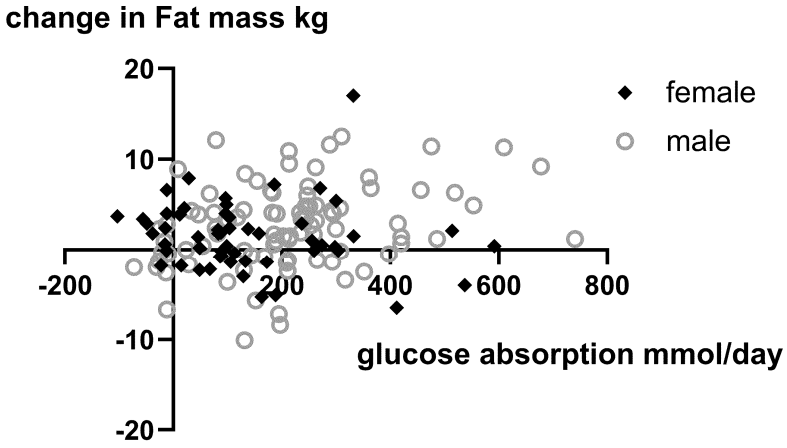

Figure 2. Spearman univariate correlation between daily glucose absorption from peritoneal dialysate and change in fat mass for both men (rho =0.32, p=0.002) and women. (rho=0.17, p=0.22).

Figure 3. Spearman univariate correlation between daily glucose absorption from peritoneal dialysate and change in fat free mass for both men (rho = -0.28, p=0.007) and women. (rho= -0.09, p=0.51).

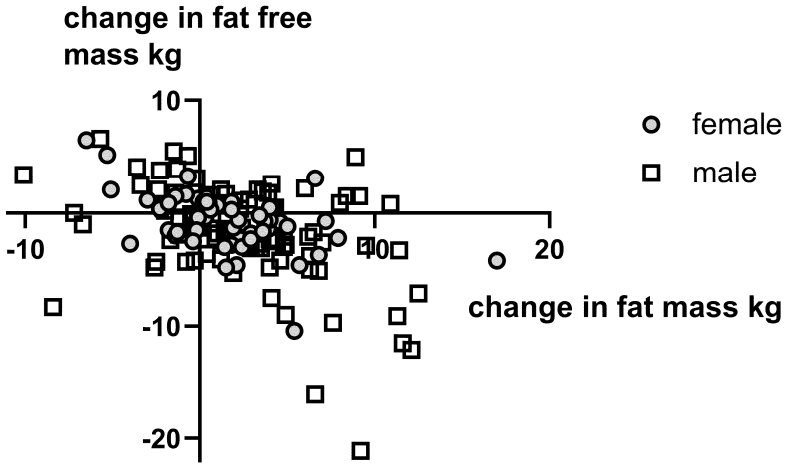

Figure 4. Spearman univariate correlation between change in fat mass and change in fat free mass for both men (rho = -0.40, p=0.001) and women. (rho= -0.46, p=0.004).

Table 1. Patient demographics. Patients divided according to below and above median daily glucose absorption. Automated peritoneal dialysis (APD), continuous ambulatory peritoneal dialysis (CAPD), weekly urea clearance (Kt/V), peritoneal dialysis (PD), ultrafiltration (UF), normalised protein appearance rate (nPNA), combined urinary urea and creatinine clearance (urine clearance), dialysate to plasma ratio (D/P), body mass index (BMI), body surface area (BSA), fat mass (FM), fat free mass (FFM), glycated haemoglobin (HbA1c), C reactive protein (CRP). * p <0.05 ** <0.01, *** 0.001 vs lower glucose absorption group

variable	All	Lower absorption	Higher absorption
Male (%)	89 (62.2)	34 (47.2)	55 (77.5) ***
Age years	61.3±14.9	62.4 ±14.0	60.2 ±15.8
Diabetic (%)	53 (37.1)	30 (41.7)	32 (45.1)
Vintage months	14 (13-15)	14 (13-15)	14 (13-15)
APD	16 (11.2)	12 (16.7)	4 (5.6)
APD+day	90 (62.1)	34 (42.2)	56 (78.9)
CAPD	37 (25.9)	26 (36.1)	11 (15.5)***
Icodextrin L/day	1.5 (1-2)	1.5(1.0-2.0)	1.8 (1.3-2.0)*
22.7 g/L glucose L	1.2 (0-4.8)	0 (0-0)	4.8 (2.0-8.0)***
Kt/V_{urine}	0.9(0.4-1.37)	1.23(0.9-2.22)	0.56(0.19-0.91)***
Kt/V_{PD}	1.23(0.92-1.45)	1.02(0.75-1.29)	1.34(1.13-1.53)***
KT/V_{total}	2.17(1.74-2.66)	2.31(1.98-3.03)	1.89(1.56-2.35)***
24-hour UF_{PD} mL	596(283-882)	498(244-800)	681(396-1032)*
nPNA g/kg/day	0.91 ±0.22	0.98 ±0.23	0.85 ±0.20***
Urine clearance mL/min	3.6(2-5.6)	5.2(3.3-8.3)	2.4(0.9-3.9)***
4 hour $D/P_{creatinine}$	0.73±0.12	0.71±0.14	0.74±0.09
BMI kg/m^2	27.0 ±4.5	26.5 ±4.9	27.5 ±4.1
BSA m^2	1.87 ±0.22	1.81 ±0.23	1.93 ±0.20**
Fat mass kg	24.8 ±10.3	24.0 ±10.3	25.5 ±10.3
Fat mass index kg/m^2	9.1±3.9	9.0 ±4.0	9.1 ±3.9
Fat Free mass kg	49.9±10.6	47.2±10.8	52.6±9.7**
Fat free mass index kg/m^2	17.9±2.4	17.4±2.5	18.4±2.2*
Change FM kg	1.8 (-0.5 to 4.4)	0.1(-1.3 to 3.7)	2.7(0.4 to 6/3)**
Change FFM kg	-1.3 (-2.9 to 1.0)	-0.6(-2.2 to 1.1)	-1.9 (-4.1 to 0.9)*
Glucose mmol/L	5.8(4.7-10.1)	6.2 (4.7-11.2)	5.7 (4.7-7.4)
HbA1c mol/mol	39.9(33.2-49.7)	39.5(34.4-49.7)	39.9(32.3-49.7)
Albumin g/L	38.7±3.5	38.9±3.4	38.5±3.6
CRP g/L	3 (1-7)	3 (1-6)	4 (1-7)
Haemoglobin g/L	109.1±16.3	110.6 ±18.5	107.5 ±13.5

Table 2. Patient demographics. Patients divided according to peritoneal dialysis (PD) modality :automated peritoneal dialysis (APD), continuous ambulatory peritoneal dialysis (CAPD), weekly urea clearance (Kt/V), ultrafiltration (UF), normalised protein appearance rate (nPNA), combined urinary urea and creatinine clearance (urine clearance), dialysate to plasma ratio (D/P), body mass index (BMI), body surface area (BSA), fat mass (FM), fat free mass (FFM), glycated haemoglobin (HbA1c), C reactive protein (CRP). * p <0.05 ** <0.01, *** 0.001 vs APD

variable	APD	APD + day fill	CAPD
Male (%)	3 (18.8)	61 (67.8)**	25 (67.6) **
Age years	58.6±17.7	60.8 ±14.2	63.6 ±15.5
Diabetic (%)	2 (12.5)	35 (38.9)	16 (43.2)
Vintage months	14.5 (14-16)	14 (13-15)	14 (13-18)
Glucose absorption mmol/day	93 (75-170)	210 (118-293)***	176 (-15 to191)
cycles	5 (4-6)	6 (5-7)	4 (1-4)
Glucose L/day	8.6 (7.6-9.6)	10.5 (9.5-13.8)	2 (0-6)
Icodextrin L/day	0 (0-0)	1.5(1.0-2.0)***	2.0 (2.0-2.0)***
22.7 g/day dextrose	0 (0-0)	1.5 (1.0-2.0)***	0 (0-2.0)***
Kt/V_{urine}	1.37(1.06-2.39)	0.68(0.29-1.05)**	1.23(0.74-2.27)**
Kt/V_{PD}	1.0(0.89-1.23)	1.33(1.09-1.53)**	0.84(0.49-1.28)**
KT/V_{total}	2.53(2.17-3.47)	2.02(1.68-2.48)*	2.15(1.82-2.86)
24-hour ultrafiltration mL	339(185-566)	672(365-1018)***	500(200-800)
nPNA g/kg/day	0.60±0.15	0.73±0.09***	0.77±0.12***
Urine clearance mL/min	5.3 (3.6-7.4)	3.0 (1.3-4.7)**	4.6 (3.0-9.0)
4 hour $D/P_{creatinine}$	0.60±0.15	0.73±0.09***	0.77±0.12***
BMI kg/m^2	24.9±4.4	27.6±4.5	26.2±4.5
BSA m^2	1.69±0.18	1.92±0.22***	1.82±0.20
Fat kg	21.4 ±9.1	26.5 ±10.7	22.0 ±9.1
Fat mass index kg/m^2	8.5±3.8	9.5 ±4.0	8.2 ±3.7
Fat Free mass kg	42.4±18.2	51.4±10.4*	49.5±10.7*
Fat free mass index kg/m^2	16.4±2.1	18.1±2.4*	18.0±2.3
Change FM kg	1.7 (-0.8 to 3.2)	2.0(-0.3 to 5.4)	1.5(-0.5 to3.5)
Change FFM kg	-1.4 (-2.3 to 1.0)	-1.5(-3.1 to 0.9)	-0.4 (-2.5 to 1.0)
Glucose mmol/L	5.1 (4.5-5.9)	6.0 (4.7-9.0)	6.4 (4.8-12.1)
HbA1c mol/mmol	36.6(32-43.2)	40.5(34.4-49.7)	40.5(32.8-56.1)
Albumin g/L	41.1±3.3	38.5±3.2	38.3±3.9
CRP mg/L	2(1-2.5)	4 (1-7)	3 (1-7)
Haemoglobin g/L	103 ±13	109 ±15	112 ±20

Table 3: Spearman univariate analysis with daily glucose absorption as dependent variable.

variable	rho	p
22.7 g/L dextrose L/day	0.69	<0.0001
Weekly urinary Kt/Vurea	-0.60	<0.0001
Combined urinary urea creatinine clearance	-0.56	<0.0001
Weekly peritoneal Kt/Vurea	0.54	<0.0001
Volume glucose peritoneal dialysate/day	0.52	<0.0001
Cycles peritoneal dialysate/day	0.35	<0.0001
Normalised protein appearance rate	-0.29	0.008
Fat free mass kg	0.26	0.007
Body surface area m^2	0.25	0.003
24-hour peritoneal ultrafiltration mL	0.19	0.023
Blood glucose mmol/L	-0.17	0.033

Table 4. Backward logistic regression model of daily glucose absorption above and below the median. Standard error (SE), 22.7 g/L glucose dialysate (22.7 g/L), weekly peritoneal urea clearance (Kt/V_{PD}),Odds ratio, confidence limits (CL). Nagelkerke r^2 =0.68

variable	β	SE of β	Wald	OR	95% CL	p
22.7g/L L/day	1.68	0.13	27.9	2.0	1.5-2.5	<0.0001
Kt/V_{PD}	2.25	0.70	10.2	9.5	2.4-37.1	0.001
Male vs female	1.56	0.58	7.1	4.8	1.5-14.9	0.008
Diabetic	-1.54	0.64	5.7	0.21	0.06-0.76	0.011

References

1. Twardowski ZJ, Nolph KD, Khanna R, Prowant BF, Ryan LP, Moore. HL, Neilson MP. Peritoneal Equilibration Test. Perit Dial Bull 1987;7:138-47
2. Dombros N, Dratwa M, Feriani M, Gokal R, Heimbürger O, Krediet R, Plum J, Rodrigues A, Selgas R, Struijk D, Verger C; EBPG Expert Group on Peritoneal Dialysis. European best practice guidelines for peritoneal dialysis. 8 Nutrition in peritoneal dialysis. Nephrol Dial Transplant. 2005;20 Suppl 9:ix28-ix33
3. NKF-DOQI CLINICAL PRACTICE GUIDELINES FOR PERITONEAL DIALYSIS ADEQUACY. Assessment of Nutritional status. AmJKidDis 2007; 30(3 Suppl 2), S125-9
4. Pajek J, Gucek A, Kveder R, Bucar-Pajek M, Kaplan-Pavlovcic S, Bren AF. Impact of dialysis duration and glucose absorption on nutritional indices in stable continuous ambulatory peritoneal dialysis patients. J Ren Nutr. 2008;18(6):503-8
5. Jakić M, Stipanić S, Mihaljević D, Zibar L, Lovcić V, Klarić D, Jakić M. The impact of glucose absorbed from dialysis solution on body weight gain in peritoneal dialysis treated patients. Lijec Vjesn. 2005;127(5-6):116-2
6. Nordfors L, Heimbürger O, Lönnqvist F, Lindholm B, Helmrich J, Schalling M, Stenvinkel P. Fat tissue accumulation during peritoneal dialysis is associated with a polymorphism in uncoupling protein 2. Kidney Int. 2000;57(4):1713-9
7. Caron-Lienert RS, Poli-de-Figueiredo CE, Figueiredo AEPL, da Costa BEP, Crepaldi C, Pizzato AC, Ferrari F, Giuliani A, Ronco C. The Influence of Glucose Exposure Load and Peritoneal Membrane Transport on Body Composition and Nutritional Status Changes after 1 Year on Peritoneal Dialysis. Perit Dial Int. 2017;37(4):458-463
8. Vasselai P, Kamimura MA, Bazanelli AP, Pupim LB, Avesani CM, da Mota Ribeiro FS, Manfredi SR, Draibe SA, Cuppari L. Factors associated with body-fat changes in prevalent peritoneal dialysis patients. J Ren Nutr. 2008;18(4):363-9
9. Choi SJ, Kim NR, Hong SA, Lee WB, Park MY, Kim JK, Hwang SD, Lee HK. Changes in body fat mass in patients after starting peritoneal dialysis. Perit Dial Int. 2011;31(1):67-73
10. Jager KJ, Merkus MP, Huisman RM, Boeschoten EW, Dekker FW, Korevaar JC, Tijssen JG, Krediet RT; NECOSAD Study Group. Nutritional status over time in hemodialysis and peritoneal dialysis. J Am Soc Nephrol. 2001;12(6):1272-9
11. Davies SJ, Woodrow G, Donovan K, Plum J, Williams P, Johansson AC, Bosselmann HP, Heimbürger O, Simonsen O, Davenport A, Tranaeus A, Divino Filho JC.

Icodextrin improves the fluid status of peritoneal dialysis patients: results of a double-blind randomized controlled trial. J Am Soc Nephrol. 2003;14(9):2338-44.

12. Cho KH, Do JY, Park JW, Yoon KW. Effect of icodextrin dialysis solution on body weight and fat accumulation over time in CAPD patients. Nephrol Dial Transplant. 2010;25(2):593-9
13. Davies SJ, Davenport A. The role of bioimpedance and biomarkers in helping to aid clinical decision-making of volume assessments in dialysis patients. Kidney Int. 2014;86(3):489-96
14. NKF-K/DOQI Clinical practice guidelines for peritoneal dialysis adequacy: Clinical practice recommendations for peritoneal dialysis adequacy Am J Kid Dis 2006; 48 [Suppl 1]: S98–S158
15. Davenport A, Willicombe MK. Hydration status does not influence peritoneal equilibration test ultrafiltration volumes. Clin J Am Soc Nephrol. 2009;4(7):1207-12
16. Fürstenberg A, Davenport A. Assessment of body composition in peritoneal dialysis patients using bioelectrical impedance and dual-energy x-ray absorptiometry. Am J Nephrol. 2011;33(2):150-6
17. Persaud J, Thomas M, Davenport A. Indirect ion selective electrode methods potentially overestimate peritoneal dialysate sodium losses. Ther Apher Dial. 2014;18(4):321-5
18. Rajakaruna G, Caplin B, Davenport A. Peritoneal protein clearance rather than faster transport status determines outcomes in peritoneal dialysis patients. Perit Dial Int. 2015; 35(2):216-21
19. Vongsanim S, Salame C, Eaton S, Grimble G, Davenport A. Differences between Measured Total Nitrogen Losses in Spent Peritoneal Dialysate Effluent and Estimated Nitrogen Losses. J Ren Nutr. 2019;29(3):243-247.
20. Davies SJ, Phillips L, Naish PF, Russell GI. Quantifying comorbidity in peritoneal dialysis patients and its relationship to other predictors of survival. Nephrol Dial Transplant. 2002;17(6):1085-92.
21. Rockwood K, Song X, MacKnight C, Bergman H, Hogan DB, McDowell I, et al. A global clinical measure of fitness and frailty in elderly people. CMAJ. 2005;173(5):489-95
22. Burkart J. Metabolic consequences of peritoneal dialysis. Semin Dial 2004; 17: 498-504
23. McCafferty K, Fan SL. Are we underestimating the problem of ultrafiltration in peritoneal dialysis patients? Perit Dial Int. 2006;26(3):349-52

24. Bazanelli AP, Kamimura MA, da Silva CB, Avesani CM, Lopes MG, Manfredi SR, Draibe SA, Cuppari L. Resting energy expenditure in peritoneal dialysis patients. Perit Dial Int. 2006;26(6):697-704
25. Davenport A. Effect of intra-abdominal dialysate on bioimpedance-derived fluid volume status and body composition measurements in peritoneal dialysis patients. Perit Dial Int. 2013;33(5):578-9
26. Davenport A. Does peritoneal dialysate affect body composition assessments using multi-frequency bioimpedance in peritoneal dialysis patients? Eur J Clin Nutr. 2013;67(2):223-5
27. Udo A, Goodlad C, Davenport A. Impact of Diabetes on Extracellular Volume Status in Patients Initiating Peritoneal Dialysis. Am J Nephrol. 2017;46(1):18-25
28. Davenport A, Willicombe MK. Does diabetes mellitus predispose to increased fluid overload in peritoneal dialysis patients? Nephron Clin Pract. 2010;114(1):c60-6
29. Little J, Phillips L, Russell L, Griffiths A, Russell GI, Davies SJ. Longitudinal lipid profiles on CAPD: their relationship to weight gain, comorbidity, and dialysis factors. J Am Soc Nephrol. 1998;9(10):1931-9
30. Wang X, Axelsson J, Nordfors L, Qureshi AR, Avesani C, Barany P, Schalling M, Heimbürger O, Lindholm B, Stenvinkel P. Changes in fat mass after initiation of maintenance dialysis is influenced by the uncoupling protein 2 exon 8 insertion/deletion polymorphism. Nephrol Dial Transplant. 2007;22(1):196-20
31. Choi SJ, Park MY, Kim NR, Hwang SD. The 24-month changes in body fat mass and adopokines in patients starting peritoneal dialysis. Perit Dial Int. 2017;37 (3):290-297
32. El-Kateb S, Sridharan S, Farrington K, Fan S, Davenport A. Comparison of equations of resting and total energy expenditure in peritoneal dialysis patients using body composition measurements determined by multi-frequency bioimpedance. Clin Nutr. 2018;37(2):646-650
33. El-Kateb S, Sridharan S, Farrington K, Davenport A. Comparison of resting and total energy expenditure in peritoneal dialysis patients and body composition measured by dual-energy X-ray absorptiometry. Eur J Clin Nutr. 2016;70(11):1337-133
34. Wright M, Woodrow G, O'Brien S, King N, Dye L, Blundell J, Brownjohn A, Turney J. Disturbed appetite patterns and nutrient intake in peritoneal dialysis patients. Perit Dial Int. 2003;23(6):550-6.
35. Hall KD. What is the required energy deficit per unit weight loss? Int J Obes (Lond). 2008;32(3):573-6