Review

Blood Purification

Blood Purif 2019;48:25–31
DOI: 10.1159/000499406

Received: January 4, 2019
Accepted: February 19, 2019
Published online: April 4, 2019

Counteracting the Metabolic Effects of Glucose Load in Peritoneal Dialysis Patients; an Exercise-Based Approach

Sana F. Khan[a] Claudio Ronco[b] Mitchell H. Rosner[a]

[a]Division of Nephrology, University of Virginia Health System, Charlottesville, VA, USA; [b]International Renal Research Institute of Vicenza, San Bortolo Hospital, Vicenza, Italy

Keywords
Peritoneal dialysis · Glucose load · Exercise · Complications

Abstract
Glucose-based peritoneal dialysis (PD) solutions are the predominantly used dialysate in PD patients. Glucose absorption has been shown to be associated with several unfavorable metabolic complications. Several studies have shown positive effects of exercise in end-stage renal disease patients. This paper provides an overview of glucose-associated metabolic complications, and proposed exercise regimens to counteract the caloric load associated with glucose absorption.

Introduction

Since the introduction of continuous ambulatory peritoneal dialysis (CAPD) in the 1970s, there has been a progressive increase in the number of end-stage renal disease (ESRD) patients undergoing peritoneal dialysis (PD) over the last 4 decades. Current data suggest that 348,000 patients receive PD worldwide, representing approximately 9% of the global ESRD population [1, 2]. Early experience with PD revealed concerns about safety and efficacy of the modality, as well as higher mortality rates compared to in-center hemodialysis (HD) [3–5]. Over time, there have been substantial improvements in the clinical application of PD and since the mid-1990s there has been a significantly larger reduction in the risk of death in patients undergoing PD compared to those treated with HD [6, 7]. More recently, several studies indicate PD and HD to have similar short-term (1–2 years) or long-term (up to 5 years) survival [6–10]. Despite improvements in dialysis survival over the years, adjusted survival for PD patients is still poor and suboptimal with 67% of patients surviving at 3 years after ESRD onset [11].

A number of studies have tried to identify PD-specific risk factors that increase the risk of death in this patient population. An area of investigation has been glucose (dextrose), which is utilized as the crystalloid osmotic agent in standard PD solutions. Clinical concerns associ-

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www.karger.com/bpu

Sana F. Khan, MD
Division of Nephrology
Box 800133, University of Virginia Health System
Charlottesville, VA 22908 (USA)
E-Mail sk4yp@virginia.edu

Table 1. Dextrose concentration (g) and caloric load (kcal) per standard bag sizes

	1.5 L		2.0 L		2.5 L		3 L		5 L	
	g	kcal	g	kcal	g	kcal	g	kcal	g	kcal
1.5%	22.5	83	30	111	37.5	139	35	130	75	278
2.5%	37.5	139	50	185	62.5	232	75	278	125	463
4.25%	63.75	236	85	315	106.25	393	127.5	472	212.5	786

Caloric load has been rounded up to the nearest kcal.

ated with the use of glucose-based PD solution include systemic metabolic effects and local biocompatibility effects on the peritoneum that over time may lead to peritoneal fibrosis. Several studies have examined the use of alternative osmotic agents (sorbitol, mannitol, xylitol, icodextrin); however, none have been shown to have a superior safety and efficacy profile compared to glucose [12]. This paper presents an overview of glucose absorption from PD solutions, its associated metabolic complications, as well as an exercise-based strategy that we hypothesize could combat the detrimental effects of glucose absorption.

Glucose Absorption during PD

Ultrafiltration in PD is a result of an osmotic or oncotic force across the peritoneal membrane. Current commercially available PD solutions contain 1 of 3 osmotic agents; glucose, icodextrin or amino acids, with only the former 2 approved by the Food and Drug Administration for use in the United States. Both amino acid and icodextrin containing solutions are approved for only one exchange in a 24-h period. Hence glucose-based solutions remain the predominantly used PD solutions.

Currently available PD solutions contain 1,360, 2,250 or 3,860 mg/dL of glucose, which correspond to 1.5, 2.5, or 4.25% dextrose monohydrate solutions respectively. Target ultrafiltration volumes are achieved by varying the instilled glucose concentration. Unfortunately, glucose is absorbed from the dialysate into the blood stream down its concentration gradient as the concentration in PD fluid is generally higher than the patient's blood glucose concentration [13]. Besides the tonicity of dialysate, the amount of glucose absorbed depends on peritoneal membrane transport characteristics, dwell time, dialysate volume, and the patient's blood glucose level [14].

Several investigators have studied glucose absorption from PD dialysate. One study ($n = 7$) observed absorption of 78.5% of peritoneal glucose load, average 117 ± 13.5 g/day [15]. Another study (41 2 L, 6-h dwells) revealed absorption of 75% of the initial intraperitoneal glucose concentration at the end of 6 h. Of the total amount of glucose absorbed, 50% occurred during the first 90 min of the dwell. Other studies suggest that 60–80% of instilled glucose is absorbed during a typical 6-h dwell. The percentage of glucose absorbed over time was almost identical for the 3 different dextrose concentration solutions [16]. The normal glucose uptake for a patient on continuous CAPD ranges from 100 to 300 g/day [17].

Based on the known glucose absorption, caloric intake from a CAPD regimen can be estimated by multiplying the amount of total glucose absorbed (60–80%) by 3.7 (conversion factor for gram to kcal). Similarly, the caloric intake from shorter automated PD dwells is estimated to be lower at 40–50% [16]. Glucose concentrations and the caloric load per standard sized bags for the 3 different solutions are listed in Table 1.

Certain studies have estimated glucose absorption in terms of calories absorbed. Peritoneal glucose energy intake has ranged between 4 and 13 kcal/kg/day [18] and 5–29 kcal/kg/day [19] in different studies. Kinetic modeling programs can also be used to predict glucose absorption and caloric intake [20].

Metabolic Complications Commonly Attributed to Glucose Absorption

While obligatory carbohydrate absorption contributes to the total energy intake of patients undergoing PD, it has been shown to result in worsened glycemic control in diabetic patients. This obligatory glucose absorption is higher in patients with faster peritoneal solute transfer characteristics. Not surprisingly, the mean 24-h blood glucose concentration has been shown to be significantly associated with dialysate glucose concentrations [21]. While glucose-based dialysate may worsen glycemic control, there is evidence of improved glycemic control using glucose-sparing solutions. In one study, substitution of a glucose-based solution with an icodextrin-based solution resulted in significant reduction in glucose absorption as

well as insulin needs [22]. Additionally another study showed that substitution of 2 daily glucose-based exchanges with one exchange each of icodextrin and amino acids is associated with significantly lower mean blood glucose when measured with a continuous glucose monitoring system [23]. A larger randomized trial with the same icodextrin and amino acid substitutions resulted in an improved HbA1c of individuals in the intervention arm [28].

There are limited data on the role of PD therapy in causing new-onset diabetes. However, one study has shown that 8% of nondiabetic patients became diabetic after initiating PD [24].

Dyslipidemia is another metabolic complication of PD and has been attributed to a combination of obligatory carbohydrate absorption and peritoneal protein loss. Dyslipidemias observed in PD patients have been well characterized. The most common abnormalities include elevated total cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides, and lipoprotein A and apolipoprotein B (apoB) levels. High density lipoprotein and apolipoprotein A1 levels are usually low. Compared with HD patients, prominent differences are the elevated apoB and LDL cholesterol levels [25]. Studies substituting one glucose-based exchange with one exchange of icodextrin showed a 5–10% reduction in total and LDL cholesterol, with no effect on serum triglycerides or very LDL cholesterol [26, 27]. Conversely, the incorporation of amino acids as well as icodextrin as part of glucose-sparing regimen showed lower triglycerides and very LDL cholesterol levels, without any significant change in total or low-density cholesterol levels [28]. The contribution of dyslipidemias to the high cardiovascular risk in PD patients remains unclear. The SHARP trial examined the effects of lipid lowering on cardiovascular events and mortality (including 496 PD patients). While the number of cardiovascular events was significantly lower in the intervention arm, there was no effect on either cardiovascular or all-cause mortality. Perhaps, the benefit from lipid lowering may not be as significant in patients with end-stage kidney disease, as compared to the general population.

Weight gain is commonly attributed, in part, to the absorption of glucose with PD therapy and has been noted in several studies [29–31]. However, weight gain on PD initiation has not been noted to be different from gain on HD initiation and may be more related to increases in appetite associated with treatment of uremia. One study revealed similar trajectories of weight gain in both PD and HD patients, with more fat gained by women on PD and diabetic patients [30]. Another study concluded that the probability of significant weight gain was more likely in patients initiating HD rather than PD [32]. Despite similar magnitude of weight gain, patients initiating PD have been noted to have a greater increase in visceral fat compared with those initiating HD. These longitudinal studies have small numbers of patients and need to be validated in larger cohorts [33–35].

Table 2. Metabolic effects associated with peritoneal glucose absorption

– Hyperglycemia
– Increased insulin need
– Dyslipidemias
– Weight gain
– Increased visceral fat
– Metabolic syndrome

Recent studies have revealed that hormones secreted by adipocytes play a role in the development of complications seen in PD patients. The proinflammatory effects of adipose tissue have been associated with development of metabolic syndrome in PD patients [36]. The role of peritoneal glucose exposure on adipocytes requires further study.

Current data on metabolic complications attributed to systemic glucose absorption are conflicting. Despite the inconclusive studies, it is plausible that obligatory glucose absorption with PD contributes to adverse cardiovascular risks as mentioned above. Thus, it would be prudent to limit exposure to hypertonic glucose solutions, given the positive effects observed in trials using glucose-sparing dialysate on various metabolic parameters. Table 2 lists a summary of metabolic effects attributed to glucose absorption.

Counteracting the Caloric Load from Glucose Absorption: An Exercise-Based Regimen

The effects of aerobic exercise have been studied extensively in HD patients. Besides improvement in physical capacity (measured by changes in maximal aerobic capacity, VO_{2max}), exercise has been shown to positively influence patients' lipid profiles, blood pressure control, and quality of life [37–41]. Data on exercise training in PD patients are limited. A study of exercise conditioning in 7 CAPD patients revealed improved quality of life, physical capacity, fasting blood glucose levels, and a trend toward increased HDL cholesterol. These patients underwent exercise conditioning 3 days a week for a duration of 12 weeks, and utilized treadmill, ski training machine,

Table 3. Weight-based caloric consumption (kcal) associated with 60 min of selected physical activities

Activities (METS)	125 lb	155 lb	185 lb	215 lb	245 lb
Stationary bike-moderate effort (4.8)	286	354	423	491	560
Elliptical trainer (5.0)	298	369	439	512	583
Yoga (2.5)	149	184	220	256	292
Pilates (3.0)	179	221	264	298	360
Rope jumping, slow pace (8.8)	524	650	753	901	1,027
Jazzercise (6.0)	358	442	528	596	720
Video exercise workouts (4.0)	238	295	351	400	453
Low impact aerobics (5.0)	298	369	439	512	583
Walking 2.8–3.2 mph, moderate pace(3.5)	202	251	308	358	408
Walking 3.5 mph, brisk pace (4.3)	249	317	405	440	502
Running, jog/walk (6.0)	358	442	528	596	720
Running, 4 mph (6.0)	358	442	528	596	720
Swimming, light effort (5.8)	345	428	511	594	677
Water aerobics (5.3)	316	391	467	543	618
Aqua jogging, moderate pace (4.5)	268	332	396	461	523
Golfing (4.8)	286	354	423	491	560
Yard work (4.0)	238	295	351	400	453
Lawn mowing (5.5)	327	406	485	563	642

and upper limb and bike ergometers [42]. In another study, ESRD patients (including 12 CAPD patients) completed thrice weekly aerobic training for 3 months on a bike ergometer, and revealed improved peak exercise capacity [43]. An evaluation of a weight reduction program in PD patients revealed significant weight loss in the majority of subjects, though the methods were via dietary control as well as exercise [44]. Additionally, a small study investigating the effects of exercise revealed significantly improved fasting glucose and 2-h post prandial blood sugar. This group of CAPD patients exercised on a stationary bike for twice-weekly 40 min sessions, and completed 16 sessions of exercise [45].

To date, there has been no study evaluating the effects of an exercise-based regimen aimed to counteract the caloric load from glucose absorption and specifically designed to balance the excess calories associated with glucose absorption from the peritoneal dialysate. We propose an exercise regimen to negate the daily glucose load from patients' individual prescriptions. As mentioned above, 60–80% and 40–50% of glucose load are absorbed via CAPD and CCPD regimens respectively. Using data shown in Table 1, the minimum daily caloric load can be calculated via Equations 1 and 2, and explained via the following examples

Equation 1 (CAPD) – Total grams dextrose × 60% × 3.7 (kcal/g conversion factor)

Equation 2 (CCPD) – Total grams dextrose × 40% × 3.7 (kcal/g conversion factor)

Example 1 – CAPD regimen, 2 L dwell volume, 4 exchanges (2 × 2.5%, 2 × 1.5%)

– Total dextrose load; (30 × 2) + (50 × 2) = 160 mg Dextrose

– Total caloric load; 160 × 0.6 × 3.7 = 355.2 kcal

Once the daily caloric load is determined based on individual patients' prescriptions, the patients can follow their exercise regimen of choice. The critical factor would be for the patient to match the caloric expenditure of the exercise with the caloric load due to the dialysate glucose. We recommend exercises that the patients can do themselves and are simple, safe, and have a low impact. Examples of caloric consumption for an average sized individual from 2 specific regimens are explained below. Physical activities detailed in Example 2 as well as Table 3 have associated METS (metabolic equivalent; measure of exercise intensity based on oxygen consumption) that can be used to calculate caloric consumption.

The 2 exercises and their associated METS are (1) stationary bicycle at 51–89 watts (4.8 METS) and (2) walking for exercise at 3.5 mph (4.3 METS). How these can be utilized to estimate caloric consumption is explained below [46].

1 MET equals oxygen consumption of 3.5 mL/kg/min. Hence, patient's individual weight can be used to estimate total oxygen consumption for a particular activity, from which calories can be calculated, given approximately 5 kcal/L of O_2. This concept is further explained via Example 2 clarified below.

Table 4. Caloric load (kcal) based on individual PD prescription (L)

	Volume, L	Caloric load/L, kcal	Total caloric load
1.5%	×	55.3	=
2.5%	×	92.3	=
4.25%	×	157.3	=

Sum of load from each percentage dextrose.

CAPD caloric absorption = total caloric load × 0.6.

CCPD caloric absorption = total caloric load × 0.4.

Volume of each percentage dextrose (L) is filled and multiplied by kcal/L to calculate total caloric load.

Sum total of caloric load from each percentage dextrose is then multiplied by 0.6 and 0.4 to calculate absorption via CAPD and CCPD respectively.

PD, peritoneal dialysis; CAPD, continuous ambulatory peritoneal dialysis.

Example 2– 72 kg male, with the caloric gain from Example 1 (355.2 kcal)

(1) Stationary bicycle at 51–89 watts (4.8 METS)

1 MET = 3.5 mL/kg/min

Oxygen consumption in mL/min = METS × body weight = 4.8 × 72 × 3.5 = 1,209.6 mL

Given approx. 5 kcal/L O_2, energy consumption = 1.2 × 5 = 6 kcal/min

Time needed to exercise at this intensity for 355 kcal = 59 min

(2) Walking for exercise at 3.5 mph (4.3 METS)

Oxygen consumption in mL/min = 4.3 × 72 × 3.5 = 1,083.6 mL

Energy consumption = 1.08 × 5 = 5.4 kcal/min

Time needed to exercise at this intensity for 355 kcal = 65 min

As explained above, for this average sized individual, approximately 1 h of exercise should be sufficient to compensate for calories gained via glucose absorption. The duration of exercise per individual patient can be varied by factoring in their caloric gains, as well as body weight. Caloric consumption associated with several activities over a range of patient weights has been detailed in Table 3. Additionally, a calculation table estimating total caloric load over a 24-h period is demonstrated in Table 4.

There are however certain caveats that need to be considered when prescribing an exercise regimen. At baseline, PD patients have been reported to have a high prevalence of sedentary lifestyle in a cohort of patients assessed by pedometers [47]. Another study reported a high prevalence of low-performance capacity among elderly patients treated with PD. Interestingly, performance level of PD patients did not appear to differ from the cohort of non-dialysis CKD patients [48]. Objectively measured physical function and ability to perform activities of daily living and self-care have been reported to be reduced across the CKD-Dialysis-Transplant spectrum [49]. A recent study evaluating physical functioning and activity levels in PD patients concluded results to be lower than reference values for age and gender in the general population, and were at the levels indicating impairment [50]. It is possible that PD patients are unable to perform the light-moderate intensity aerobic exercise regimen proposed by our program. Before the benefits of the regimen can be ascertained, patients may need a gradual conditioning regimen to prepare them for exercise. Obviously, other limitations due to osteoarthritis, peripheral vascular disease and coronary disease will need to be considered as well.

The duration of exercise training before its effects on glucose-metabolism are apparent remains uncertain. Prior studies on exercise in PD patients have ranged from 2 weeks to 1-year timeframe. Low exercise capacity and physical inactivity have been identified as prognostically important for cardiovascular disease and all-cause mortality in patients across the CKD spectrum. Additionally, performance on the 6-minute walk test has recently been shown to correlate with mortality and technique failure in a small population of ambulatory PD patients. Despite the uncertainty on the duration of an exercise program, it seems logical to initiate interventions to combat physical dysfunction and inactivity in all PD patients.

Future Studies

While the implementation of a moderate exercise program designed to address the patient's excess exposure to peritoneal dialysate glucose makes intuitive sense, this approach requires further study to investigate whether cardiovascular and mortality benefits accrue with exercise. However, this simple, inexpensive and low-impact program can be easily adapted to any home dialysis unit and as such has great promise for improving outcomes with very little downside.

Disclosure Statement

The authors declare that they have no conflicts of interest to disclose.

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