Peritoneal Dialysis

CKJ Review

Strategies for preserving residual renal function in peritoneal dialysis patients

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Abstract

Although there have been many advancements in the treatment of patients with chronic kidney disease (CKD) over the last 50 years, in terms of reducing cardiovascular risk, mortality remains unacceptably high, particularly for those patients who progress to stage 5 CKD and initiate dialysis (CKD5d). As mortality risk increases exponentially with progressive CKD stage, the question arises as to whether preservation of residual renal function once dialysis has been initiated can reduce mortality risk. Observational studies to date have reported an association between even small amounts of residual renal function and improved patient survival and quality of life. Dialysis therapies predominantly provide clearance for small water-soluble solutes, volume and acid-base control, but cannot reproduce the metabolic functions of the kidney. As such, protein-bound solutes, advanced glycosylation end-products, middle molecules and other azotaemic toxins accumulate over time in the anuric CKD5d patient. Apart from avoiding potential nephrotoxic insults, observational and interventional trials have suggested that a number of interventions and treatments may potentially reduce the progression of earlier stages of CKD, including targeted blood pressure control, reducing proteinuria and dietary intervention using combinations of protein restriction with keto acid supplementation. However, many interventions which have been proven to be effective in the general population have not been equally effective in the CKD5d patient, and so the question arises as to whether these treatment options are equally applicable to CKD5d patients. As strategies to help preserve residual renal function in CKD5d patients are not well established, we have reviewed the evidence for preserving or losing residual renal function in peritoneal dialysis patients, as urine collections are routinely collected, whereas few centres regularly collect urine from haemodialysis patients, and haemodialysis dialysis patients are at risk of sudden intravascular volume shifts associated with dialysis treatments. On the other hand, peritoneal dialysis patients are exposed to a variety of hypertonic dialysates and episodes of peritonitis. Whereas blood pressure control, using an angiotensin-converting enzyme inhibitor (ACEI) or angiotensin receptor blocker (ARB), and low-protein diets along with keto acid supplementation have been shown to reduce the rate of progression in patients with earlier stages of CKD, the strategies to preserve residual renal function (RRF) in dialysis patients are not well established. For peritoneal dialysis patients, there are additional technical factors that might aggravate the rate of loss of residual renal function including peritoneal dialysis prescriptions and modality, bio-incompatible dialysis fluid and over ultrafiltration of fluid causing dehydration. In this review, we aim to evaluate the evidence of interventions and treatments, which may sustain residual renal function in peritoneal dialysis patients.

Keywords: biocompatible dialysate ACEI; peritoneal dialysis; residual renal function

Importance of RRF in peritoneal dialysis

Peritoneal dialysis technique survival varies throughout the world, depending upon access to transplantation and haemodialysis, and centre practices [1]. Besides loss of patients to transplantation, peritonitis remains the commonest cause for transfer to haemodialysis in many countries [2], and due to the relatively high turnover of peritoneal dialysis patients, it was only in the 1990s that reports of...
the importance of maintaining residual renal function (RRF) started to appear [3]. Maiorca et al. reported a 50% reduction in mortality in peritoneal dialysis (PD) patients with RRF [4]. These encouraging results were supported by later larger prospective observational studies, with Diaz-Buxo et al., reporting that residual renal creatinine clearance (CrCl) was strongly associated with PD patient survival, whereas peritoneal clearance did not affect outcome. Moreover, they observed in their cohort of some 2686 PD patients, a dose response association between RRF and PD patient survival, with each CrCl 5 L/week/1.73 m² increase in renal creatinine clearance associated with a 10% decrease in mortality, whereas there was no association between peritoneal CrCl and mortality [5].

Similarly, Rocco et al. reported that for each 10 L/week/1.73 m² increase in renal CrCl there was a 40% reduced risk for death and also that for each increase in weekly renal Kt/Vurea of 0.1 there was a 12% reduction in the risk for death from a multicentre prospective cohort study of 1446 prevalent PD patients [6]. Once again there was no effect of peritoneal solute clearances on survival. These findings were not limited to North America or Europe [7, 8], with observational reports from Hong Kong [9] and Turkey also confirming that for every 1 mL/min increase in residual GFR mortality risk reduced by 35–47% [10, 11]. These studies emphasized the fact that the residual renal clearance may have greater beneficial effects than comparable peritoneal small solute clearance and as such these clearances are not simply equivalent. This led to the reanalysis of the CANUSA study [12], the landmark multicentre prospective cohort of 680 incident PD patients in Canada and USA, which reported that for each increment of residual renal GFR of 5 L/week/1.73 m² there was a 12% reduction in the risk for death and that for each 250 mL increase in urine volume there was a 36% decreased risk for death. Once again neither peritoneal small solute clearances nor peritoneal ultrafiltration volume were associated with patient survival. Subsequent secondary analysis of ADEMEX study additionally confirmed an advantage for RRF on mortality [13]. This cornerstone multicentre prospective randomized controlled trial of 965 Mexican PD patients, reported that for each increase in RRF of CrCl 10 L/week/1.73 m² was associated with an 11% decrease in mortality, and an increase in renal Kt/Vurea of 0.1 a 6% decrease in mortality. More recently, additional studies from the Netherlands, Sweden, Australia and New Zealand have all confirmed the importance of RRF on mortality in PD patients (Table 1) [14–16]. In addition these studies all reported additional benefits for patients with preserved RRF, ranging from improved quality of life to reduced inflammatory markers [14, 15].

Although the evidence from these large observational and interventional trials is strongly weighted to an association between preservation of residual renal function and improved patient survival, they do not prove a causal effect. One potential confounder to all these studies is one of lead-time bias, in that patients with greater residual renal function may have initiated dialysis at a relatively earlier time than those with lower residual renal function. Similarly, some patients with CKD may have been started on peritoneal dialysis after an episode of acute kidney injury, followed by some recovery of RRF.

**Measurement of residual renal function**

Simply estimating RRF by measuring urine volume is inaccurate in patients with CKD [17]. Although the clearance of inulin, isotopes and radiocontrast agents (51chromium ethylenediaminetetra-acetic acid (EDTA) and iothalamate) are more accurate for determining residual renal function in patients with CKD than urine collections [18], these add costs and are impractical for routine clinical practice. As such, most centres use 24-h urine collections, and as urinary urea falls in CKD and underestimates inulin clearance, and conversely the relative ratio of tubular secreted to glomerular filtered creatinine increases urinary creatinine [19, 20], current guidelines advocate calculating the mean of creatinine and urea clearance, and then normalizing clearance to a body surface area of 1.73 m² [21]. However dialysis patients may suffer from sarcopenia, and as such changes in body composition [22, 23].

However, both urea and creatinine are influenced by dietary protein intake, particularly meat, and creatine production depends upon both hepatic synthetic function

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**Table 1. Summary of studies reported beneficial of RRF on mortality**

| Reference (year) | Study design | Number, characteristics and modality of subjects | Measurement of RRF | RR or OR of mortality per increase of RRF (CI or P-value) |
|------------------|-------------|-----------------------------------------------|-------------------|------------------------------------------------------|
| Maiorca et al. (1995) [4] | 3-year prospective single centre | Prevalent 68 CAPD and 34 HD | GFR 10 L/week/1.73 m² | 0.4 (P < 0.001) |
| Díaz-Buxo et al. (1999) [5] | 1-year prospective single centre | Prevalent 2686 CAPD or CCPO | Renal CrCl 10 L/week/1.73 m² | 0.89 (P = 0.003) |
| Rocco et al. (2000) [6] | 7-month prospective multicentre | Prevalent 1446 CAPD or CCPO | Renal CrCl 10 L/week/1.73 m² | 0.6 (0.4–0.8) |
| Szeto et al. (2000) [10] | 3-year prospective single centre | Prevalent 270 CAPD | GFR 1 mL/min/1.73 m² | 0.65 (0.65–0.94) |
| Ates et al. (2001) [11] | 3-year prospective single centre | Incident 125 CAPD | GFR 1 mL/min/1.73 m² | 0.53 (0.31–0.92) |
| Bargman et al. (2001) [12] | 2-year prospective multicentre | Prevalent 680 CAPD | GFR 5 L/week/1.73 m² | 0.88 (0.83–0.94) |
| Panigagua et al. (2002) [13] | 2-year multicentre randomized controlled trial | Incident 965 CAPD | Urine volume > 250 mL/day | 0.64 (0.51–0.8) |
| Termorshuizen et al. (2003) [14] | 3-year prospective multicentre | Incident 413 CAPD | Renal Kt/V 0.1 unit | 0.94 (P = 0.01) |
| Chung et al. (2003) [15] | 2-year retrospective | Incident 117 CAPD | GFR 1 mL/min/1.73 m² | 0.88 (0.79–0.99) |
| Szeto et al. (2004) [3] | 5-year prospective single centre | Prevalent 270 CAPD | GFR 1 mL/min/1.73 m² | 0.79 (0.62–0.99) |
| Rumpsfeld et al. (2009) [16] | 3-year retrospective | Incident 2436 CAPD or APD | GFR 10 L/week/1.73 m² | 0.93 (P = 0.01) |
and muscle mass and physical activity, and changes in intestinal bacteria flora alter urea and create gastrointestinal losses [24]. As such, these factors add potential confounders when reviewing serial measurements of RRF from CKD5d patients over time.

The commonest method for estimating creatinine remains the colourimetric Jaffe-based reaction. In kidney disease, chromogens accumulate which can interfere with this assay [25], and as such creatinine estimations vary between laboratories. Enzymatic methods of creatinine measurement, which are less affected, are more reliable. In addition to these technical aspects which affect measurement of RRF, urine volumes and urinary urea, creatinine and protein vary in 24-h urine collections in CKD patients not only consequent upon hydration status but also on patient compliance with completeness of the collection [25]. Although 24-h urine collections remain the standard method to determine RRF in clinical practice, there is not only interpatient and interlaboratory variation but also intrapatient variability (Figure 1).

The effect of the original cause of kidney disease on loss of residual renal function in the PD patient

The original cause of kidney disease can certainly have an impact on CKD progression, for example, most CKD5d patients with antiglomerular basement membrane disease initiate dialysis virtually anuric whereas children with nephronophthisis may be polyuric. Haynes et al. reported an annual rate of decline in residual renal function of 3.8 ± 2.5, 2.5 ± 4.8 and 1.9 ± 3.6 mL/min/1.73 m² for patients with cystic kidney disease, diabetic kidney disease and glomerulonephritis, respectively [26], and Liao et al. also noted that PD patients with diabetic nephropathy had a more rapid progressive loss of RRF [27]. However, these observations were not supported by USRDS data [28], although this study may have been confounded by including haemodialysis patients and so introducing other factors such as repeated intradialytic hypotensive episodes [29]. More recently, another study from Hong Kong reported that patients with proteinuria renal diseases were more likely to have a faster loss of RRF [30] as were those with peripheral and cardiovascular disease [31] and patients initiating PD with less RRF [30]. Loss of RRF in returning kidney transplant patients may vary with centre practices, in terms of immunosuppressive policy and reducing or stopping these medications when starting PD.

As underlying primary renal disease, and baseline GFR at PD initiation appear to have a major effect on determining loss of RRF, these factors should be considered when designing prospective interventional studies designed to preserve RRF.

Strategies for preserving RRF in patients with progressive CKD

Dietary intervention

Increased protein intake increases both the glomerular filtration rate and increases renal tubular acid excretion in the normal kidney. As hyperfiltration and increased renal tubular work load to maintain acid-base homeostasis have both been proposed as mechanisms for continued renal injury, protein restriction may potentially reduce the rate of loss of RRF. The potential benefits of dietary protein restriction (0.58 g/kg/day versus a normal dietary protein intake of 3.3 g/kg/day) to slow the progression of chronic kidney disease (CKD) were reported in the Modification of Diet in Renal Disease (MDRD) study [32], which demonstrated that a low protein diet had a modest effect when compared with blood pressure control in patients with CKD stages 3–4 (eGFR 25–55 mL/min/1.73 m²). Follow-up suggested that there may have been a continuing effect, predominantly for those with diabetic kidney disease [33]. In contrast, very low protein diets with keto acid supplements (protein intake 0.28 g/kg/day and ketoacids 0.28 g/kg/day) did not reduce progression in patients with CKD stage 4–5 (eGFR 13–24 mL/min/1.73 m²) and were associated with increased mortality when compared with those on a low-protein diet [34]. There are limited data in PD patients, although a small single centre trial reported that RRF was better maintained in incident PD patients with a urine output ≥800 mL/day or an eGFR ≥2 mL/min/1.73 m², over 12 months prescribed a low-protein diet with supplemental ketoacids (protein intake 0.6–0.8 g/kg/day with keto acids 0.12 g/kg/day) versus a low-0.6–0.8 g/kg/day and a high-protein diet group 1.0–1.2 g/kg/day [35]. Reducing dietary protein intake reduces serum creatinine, but how this affects measurement of residual renal function in patients with CKD5d is unknown, as it may increase the ratio of creatinine secreted by the tubule compared to that filtered thus giving a ‘higher’ creatinine-based estimate of RRF. Similarly it is unknown whether reducing dietary protein intake affects gastrointestinal creatinine loss. In addition, none of these studies assessed dietary sodium or phosphate intake, which are linked to dietary protein ingestion, and lower protein diets supplemented with keto acids would have been expected to contain lower sodium and phosphate content. Thus, although an observational study reporting faster loss of RRF in PD patients with higher dietary protein intake [36] may have been due to greater protein intake, it may have been confounded by higher dietary sodium and phosphate intake.

Effects of blood pressure control and RRF

The protective effects of blood pressure control to slow the progression of CKD are the basis of medical management. However, the role of blood pressure control in preserving RRF in PD patients remains inconclusive. Moist et al. reported

Fig. 1. Bland Altman plot showing variation in glomerular filtration rate calculated from sequential 24 h urine collections in 100 peritoneal dialysis patients.
Effects of renin-angiotensin-aldosterone system blockage

Although angiotensin converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) have been convincingly demonstrated to reduce the rate of progression and proteinuria in CKD patients [38], it is unclear whether they have a benefit in the PD patient [39]. Although ACEIs and ARBs improve survival in patients with chronic heart failure they reduce renal function [40], and similarly in PD patients, any potential benefit may be abrogated by an increased risk of hypotension and acute kidney injury [41]. The results of observational studies have been mixed, with a large retrospective study from USRDS on incident and prevalent PD reporting ACEIs had a protective effect on RRF [28], whereas a study of 160 incident PD patients from Australia, and 451 from the Netherlands showed no benefit [36, 42], although more diabetics were treated with ACEIs in the latter study. A recent observational study reported a small protective effect for ACEIs, but when corrected for other factors showed no statistical advantage for ACEIs [30]. Two small randomized trials have reported better preservation of RRF with ACEIs/ARBs. First, Li et al. studied 60 PD patients and reported the rate of decline in RRF over 12 months with ramipril was 2.07 mL/min per 1.73 m² versus 3.0 mL/min per 1.73 m² for the control group, although there was no difference in RRF between the groups at 3, 6 and 9 months, and the only difference was at the end of the study when a number of patients had dropped out and some had stopped taking ramipril due to side effects. Interestingly, the hazard ratio for anauria was higher in the ramipril-treated group at 3, 6, 9 months, which may be explained by the haemodynamic side effects of ACEIs [43]. Suzuki et al. reported on 34 patients randomized to valsartan or other antihypertensives and the ARB group had lower loss of RRF over 2 years from 3.2 ± 0.3 to 4.3 ± 0.7 mL/min/1.73 m² compared with 5.9 ± 0.5 to 2.8 ± 0.4 mL/min/1.73 m² in the control group [44].

Unexpectedly, RRF improved after ARB administration and was higher at 6 months than prior to starting ARBs, suggesting that some patients had regained RRF after an acute decline which had initiated starting PD treatment. Neither study showed any effect of ACEI/ARB on proteinuria. A small prospective trial showed no difference between ARBs and ACEIs on RRF [45].

More recently, a systematic review from the Cochrane library reported that ACEIs or ARBs may provide some protection in preserving RRF in PD patients, but did not reduce proteinuria. However, as the number of studies and quality of studies, in terms of potential confounders was markedly limited, no recommendation that ACEIs/ARBs should be the antihypertensive agents of choice for PD patients could be made [46]. On the other hand ACEIs/ARBs did not increase serum potassium, although the combination of ACEIs and ARBs may potentiate hyperkalaemia and oliguria in PD patients [47].

The effect of loop diuretics on RRF

In clinical practice, diuretics are commonly prescribed to PD to aid volume control, but hypovolaemia may lead to acute kidney injury and loss of RRF. Observational studies have either reported no effect on RRF [28, 36], or a loss of RRF [26, 30]. On the other hand, Van Olden et al. observed that in the short term, high dose furosemide increased both free water and sodium excretion but without affecting urea or creatinine clearance [48]. In a randomized trial, Medcalf et al. compared the effect of 250 mg/day of furosemide over 12 months in 61 incident PD patients [49] and showed that although treatment with furosemide improved fluid balance and increased urine volume and sodium excretion there was no benefit on preserving RRF. As such there is no convincing data that loop diuretics, such as furosemide maintain RRF, whereas they increase urine output and sodium excretion and as such may benefit volume overloaded patients.

Effects of peritoneal dialysis modality and RRF

Around the world, the proportion of patients treated by continuous ambulatory peritoneal dialysis (CAPD) and automated peritoneal dialysis overnight cyclers (APD) varies markedly. There has been a debate as to whether APD therapy leads to earlier loss of RRF. APD patients are generally exposed to higher glucose dialysates compared with CAPD, and glucose exposure has been reported to be associated with faster loss of RRF [36]. In addition, blood pressure tends to fall when peritoneal dialysate is drained out and then increases during infill, and it has been hypothesized that these changes in blood pressure and cardiac filling could predispose to renal hypoperfusion and earlier loss of RRF [50]. Hiroshige and Hufnagel were the first to report more rapid loss of RRF with APD in small single-centre series [51, 52]. However, others reported no difference between the two modalities in small trials [53, 54] and large observational databases and registries [26, 28, 39, 53, 55] (Table 2). More recently, registry data from the NECOSAD study reported a higher risk for loss of RRF with APD, particularly in the first year of treatment, with an adjusted hazard ratio 2.66 (confidence interval 1.60–2.44) [65]. Apart from the NECOSADS study [65], nearly all of the other studies are potentially confounded by patient selection bias and underlying original kidney disease, as generally the older more comorbid patients were treated by CAPD [54]. APD therapy using high glucose dialysates may potentially increase the risk of hypovolaemic episodes and renal ischaemia, and as such lead to an earlier loss of RRF.

Effects of biocompatible peritoneal fluid and RRF

Conventional peritoneal dialysis fluids are hypertonic and acidic, containing lactate as a base equivalent and glucose degradation products (GDPs). The newer neutral pH, lower GDP PD fluids, may better preserve RRF as they may cause less intraperitoneal inflammation, and so reduce peritoneal ultrafiltration and fluid losses [55]. This concept is supported by a short-term European study which reported greater urine volume and both urinary urea and creatinine clearance with the neutral pH low GDP glucose containing dialysates [66]. These beneficial effects of neutral pH dialysates were confirmed by a number of clinical trials, which reported better preservation of RRF with the less biocompatible dialysates [66],...
extracellular water (ECW) [84], there have been concerns that it may lead to dehydration and loss of RRF [85]. One randomized study measuring body composition and ECW reported that icodextrin reduced ECW, but also reduced urine output and GFR [85]. Only one small single-centre study reported that icodextrin usage helped preserve RRF [86], whereas five other studies showed no effect [87–91] (Table 4). As such, a recent Cochrane meta-analysis concluded that whereas icodextrin increased ultrafiltration compared with a standard 22.7 g/L glucose exchange, there was no effect on RRF [83]. However, as icodextrin can lead to a reduction in ECW, patients could potentially be at increased risk of dehydration and acute kidney injury, as dehydration is linked to loss of RRF [36].

**Effects of volume status and RRF**

Intravascular volume depletion has been widely accepted as a cause of loss of RRF in PD patients [36]. However, many studies using bioimpedance techniques have reported that PD patients generally have an increased ECW volume [92]. Although faster transporters may be expected to potentially be at greater risk of hypervolaemia due to the more rapid fall in the osmotic glucose gradient, cross-sectional studies have not reported any differences in ECW volume with transporter status in healthy APD outpatients and CAPD outpatients using 7.5% icodextrin [93].

Many clinicians err on the side of volume expansion for PD patients in the belief that this will help maintain RRF. Studies using the less bioincompatible PD dialysates reported lower peritoneal ultrafiltration, and so the question

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### Table 2. Summary of studies reported effect of dialysis modality on RRF

| Reference (year) | Study design | Subject characteristics | Favour CAPD | Details |
|------------------|--------------|-------------------------|------------|---------|
| Hiroshige et al. (1996) | 6-month prospective | Prevalent 8 NIPD, 5 CCPD, 5 CAPD | Yes | Rate of change of RRF in $-0.29$ (NIPD) versus $-0.34$ (CCPD) mL/min/month |
| Rodríguez et al. (1998) | 3-year prospective | Prevalent 25 CAPD, 20 APD | No | |
| Hufnagel et al. (1999) | 18-month prospective | Incident 6 NIPD, 12 CCPD, 18 CAPD | Yes | Rate of change of RRF in $-0.26$ (APD) versus $-0.13$ (CAPD) mL/min/month |
| Bro et al. (1999) | 6-month randomized controlled trial | Prevalent 13 CAPD, 12 APD | No | |
| Moist et al. (2000) | 3-year retrospective | Incident 722 CAPD, 310 APD | No | |
| De Fijter et al. (2000) | 2-year randomized controlled trial | Incident 13 CCPD, 11 APD | No | |
| Gollard et al. (2000) | 1-year prospective | Incident 11 CAPD, 9 APD | No | |
| Singhal et al. (2000) | 4-year prospective | Incident 211 CAPD, 31 APD | No | |
| Holley et al. (2001) | 9-year retrospective | Incident 11 CAPD, 9 APD | No | |
| Jansen et al. (2002) | 1-year prospective | Incident 243 PD subjects | No | |
| Hidaka et al. (2003) | 6-year prospective | Incident 27 CAPD, 7 APD | Yes | Approximate time to decrease 50% of RRF in CAPD is 15 months versus APD 4 months, $P < 0.001$ |
| Johnson et al. (2003) | 6-year prospective | Incident 134 CAPD, 12 APD | No | |
| Rodríguez-Carmona (2004) | 1-year prospective | Incident 53 CAPD, 51 APD | Yes | Hazard ratio of APD versus CAPD $= -1.2 (-2.25$ to $-0.15, P = 0.02)$ |
| Rabindranath (2007) | Systematic review of 3 RCT | 49 PD subjects | No | |
| Liao (2009) | 10-year prospective | Incident 188 CAPD, 82 APD | No | |
| Su et al. (2010) | 9-year retrospective | Prevalent 140 CAPD, 32 APD | No | |
| Cnossen et al. (2010) | 7-year retrospective | Incident 179 CAPD, 441 APD | No | |
| Balasubramanian et al. (2011) | 5-year retrospective | Incident 178 CAPD, 13 APD | No | |
| Michels et al. (2011) | 3-year retrospective | Incident 505 CAPD, 78 APD | Yes | Higher risk of loss of RRF in APD compared to CAPD in first year of treatment (adjusted hazard ratio $2.66, CI 1.66$–$4.44)$ |
arose as to whether any benefit in maintaining urine volume or RRF was secondary to prevention of dehydration, rather than any effect of the dialysate per se. On the other hand, sustained hypervolaemia will result in hypertension, left ventricular hypertrophy [94] and may lead to an increased risk for cardiovascular mortality. In a cross-sectional study of 550 prevalent stable PD patients which defined hypervolaemic status, as ratio of ECW to TBW measured by multifrequency electrical bioimpedance assessments (MFBIA), urine output was lower in hypervolaemic status, as ratio of ECW to TBW measured by multifrequency electrical bioimpedance assessment (MFBIA). Therefore, many are volume expanded [97]. McCafferty et al. examined the association of annual measurement of MFBIA and loss of RRF in 237 prevalent PD patients [98] and reported that there were no differences in the change in RRF with respect to absolute or relative changes in ECW/TBW ratio. Importantly, this study showed that maintaining a hypervolaemic state did not preserve RRF, and this needs to be confirmed by a prospective blinded study.

Effects of nephrotoxic insults and RRF

Nephrotoxic agents such as non-steroidal anti-inflammatory drugs, aminoglycoside antibiotics and radio-contrast iodine are recognized to increase the risk of acute kidney

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**Table 3. Summary of studies reported effect of biocompatible peritoneal solution on RRF**

| Reference (year) | Study design | Subject characteristics | Favour biocompatible solution | Details |
|------------------|--------------|-------------------------|-------------------------------|---------|
| Feriani et al. (1998) [76] | 6-month randomized controlled trial | Prevalent 33 lactate base, 36 bicarbonate base | No | Renal CrCl and urea clearance increase when using balance solution and decrease when using standard solution |
| Coles et al. (1998) [77] | 2-month randomized controlled trial | Prevalent 3 arms, 19 lactate base, 20 bicarbonate base | No | |
| Traneus et al. (2000) [78] | 1-year randomized controlled trial | Prevalence 106 CAPD, 70 bicarbonate/lactate, 36 lactate | No | |
| Rippe et al. (2001) [69] | 2-year randomized controlled trial | Prevalent 40 conventional, 40 neutral pH dialysate | No | |
| Williams et al. (2004) [66] | 6-month randomized crossover | Prevalent 86 CAPD subjects | Yes | |
| Montenegro et al. (2006) [79] | 1-year randomized crossover | Incident 36 CAPD, 18 (lactate base), 18 (bicarbonate base) | Yes | GFR decline in lactate base group, but preserved in bicarbonate group |
| Szeto et al. (2007) [70] | 1-year randomized controlled trial | Incident 25 conventional, 25 neutral | No | |
| Fan et al. (2008) [71] | 1-year randomized controlled trial | Incident 61 CAPD or APD for conventional fluid, 57 CAPD or APD for neutral fluid | No | |
| Choi et al. (2008) [74] | 1-year randomized controlled trial | Prevalent 104 CAPD, 51(neutral), 53 (conventional) | No | |
| Weiss et al. (2009) [80] | 6-month prospective crossover | Prevalent 53 CAPD | Yes | Improvement of GFR when using bicarbonate base solution |
| Pajek et al. (2009) [74] | 6-month prospective crossover | Prevalent 26 CAPD | No | |
| Haag-Weber et al. (2010) [81] | 18-month randomized controlled | Prevalent 69 CAPD, 43 (neutral), 26 (conventional) | Yes | Monthly RRF change faster in conventional group, −4.3% versus −1.5% (P = 0.04) |
| Boja et al. (2011) [72] | 2-year prospective | Incident 20 standard, 13 balance fluid | No | |
| Johnson et al. (2012) [73] | 2-year randomized controlled trial | Incident 93 conventional, 92 balance fluid | No | |
| Kim et al. (2012) [82] | 2-year randomized controlled trial | Incident 91 CAPD, 48 (balance), 43 (conventional) | Yes | Residual renal function significantly higher in balance solution at the end of study |
| Cho et al. (2013) [76] | 1-year randomized controlled trial | Incident CAPD, 32 (balance), 28 (conventional) | No | |

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**Table 4. Summary of studies reported effect of icodextrin peritoneal solution on RRF**

| Reference (year) | Study design | Subject characteristics | Favour icodextrin solution | Details |
|------------------|--------------|-------------------------|----------------------------|---------|
| Posthuma et al. (1997) [87] | 2-year randomized controlled trial | Prevalent, CCPD, 11 (icodextrin), 10 (lowest glucose) | No | |
| Plum et al. (2002) [88] | 3-month randomized controlled trial | Prevalent, APD, 20 (icodextrin), 19 (2.27% glucose) | No | |
| Konings et al. (2003) [85] | 4-month randomized controlled trial | Prevalent, CAPD and CCPD, 22 (icodextrin), 18 (glucose) | No | GFR significantly decrease in icodextrin treated group, but maintain in control group |
| Adachi et al. (2006) [86] | 2-year retrospective | Prevalence case matched control APD, 10 (icodextrin), 12 (glucose) | Yes | GFR significantly decrease in control group, but maintain in icodextrin treated group |
| Tokatori et al. (2011) [90] | 2-year randomized controlled trial | Incident, CAPD and APD, 21 (icodextrin), 20 (glucose) | No | |
Preservation of RRF is an important determinant of both PD technique and patient survival. However, loss of RRF is dependent upon the primary renal disease and patient comorbidities and is affected by lead-time bias in terms of when patients initiate PD and also whether starting PD an episode of acute kidney injury, which is then followed by partial recovery of RRF. As there are significant errors in measuring RRF in PD patients, the question arises as to whether the effects of treatments designed to preserve RRF can be truly assessed above the background variation in RRF and patient risk factors. As such, there is little if any current convincing evidence to confirm the effect of strict blood pressure control or support the use of ACEI/ARBs compared with other antihypertensive medicines or less bioincompatible peritoneal dialysates in preserving RRF. Avoidance of dehydration and episodes of acute kidney injury associated with peritonitis appear important in preserving RRF, but on the other hand deliberately keeping PD patients overhydrated, as assessed by bioimpedance [106] does not appear to preserve RRF. Further carefully designed large scale prospective studies are warranted to prove the benefits of drug and other interventions to preserve RRF.

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