APD versus CAPD in high transporters

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Abstract

Background. Automated peritoneal dialysis (APD) is widely recommended for the management of high transporters by the International Society of Peritoneal Dialysis (ISPD), although there have been no adequate studies to date comparing the outcomes of APD and continuous ambulatory peritoneal dialysis (CAPD) in this high risk group.

Methods. The relative impact of APD versus CAPD on patient and technique survival rates was examined by both intention-to-treat (PD modality at Day 90) and ‘as-treated’ time-varying Cox proportional hazards model analyses in all patients who started PD in Australia or New Zealand between 1 April 1999 and 31 March 2004 and who had baseline peritoneal equilibration tests confirming the presence of high peritoneal transport status.

Results. During the study period, 4128 patients commenced PD. Of these, 628 patients were high transporters treated with CAPD. APD-treated high transporters were more likely to be younger and Caucasian, and less likely to be diabetic. On multivariate intention-to-treat analysis, APD treatment was associated with superior survival [adjusted hazard ratio (HR) 0.56, 95% confidence interval (CI) 0.35–0.87] and comparable death-censored technique survival (HR 0.88, 95% CI 0.64–1.21). Superior survival of high transporters treated with APD versus CAPD was also confirmed in supplemental as-treated analysis (HR 0.72, 95% CI 0.54–0.96), matched case-control analysis (HR 0.60, 95% CI 0.36–0.96) and subgroup analysis of high transporters treated entirely with APD versus those treated entirely with CAPD (HR 0.29, 95% CI 0.14–0.60). There were no statistically significant differences in patient survival or death-censored technique survival between APD and CAPD for any other transport group, except for low transporters, who experienced a higher mortality rate on APD compared with CAPD (HR 2.19, 95% CI 1.02–4.70).

Conclusions. APD treatment is associated with a significant survival advantage in high transporters compared with CAPD. However, APD treatment is associated with inferior survival in low transporters.

Keywords: automated peritoneal dialysis; continuous ambulatory peritoneal dialysis; outcomes; patient survival; peritoneal equilibration test
Introduction

Over the last decade, there has been an increasing appreciation that peritoneal membrane transport characteristics play a crucial role in determining the morbidity, mortality and management of peritoneal dialysis (PD) patients [1–5]. Patients with high peritoneal permeability (high transporters) have been shown to have substantially increased risks of death and technique failure, in spite of their more rapid diffusive clearance of urea and creatinine [1,5]. Risks of death and technique failure, in spite of their more rapid diffusive clearance of urea and creatinine [1,5]. This increased risk has been attributed at least partly to rapid clearance of the glucose-associated osmotic gradient across the peritoneal membrane leading to ultrafiltration failure and fluid overload [3,4]. There is evidence that patients with symptomatic fluid retention are 3.7 times more likely to be high than low transporters [9]. Modelling studies suggest that ultrafiltration in high transporters should be maximized by prescription of short-dwell therapies, such as automated peritoneal dialysis (APD) [2]. Consequently, the International Society of Peritoneal Dialysis (ISPD) Ad Hoc Committee on Ultrafiltration Management in Peritoneal Dialysis strongly recommends APD for the treatment of high transporters with impaired net ultrafiltration [2].

In spite of these recommendations, the evidence supporting the use of APD to improve clinical outcomes in high transporters is extremely limited. One prospective, open-label, randomized, multicentre controlled trial of APD versus CAPD in 25 prevalent PD patients who were high or high-average transporters observed no significant changes in net ultrafiltration (APD 1092 ± 442 versus CAPD 1190 ± 343 ml/day) and was not statistically powered to evaluate survival outcomes [10]. A subsequent meta-analysis of three randomized controlled trials of APD versus CAPD involving 139 patients did not find any differences in patient or technique survival, but was inadequately powered to assess these outcomes and did not perform subgroup analysis in high transporters [11]. It has been argued that APD is beneficial in high transporters based on the fact that an increased mortality risk in high transporters has only been observed in patients treated with CAPD and not in those treated with APD [1,5]. However, the results are conflicting as some studies have not reported an increased risk of death in high transporters treated with CAPD [12–14], while other investigators have observed significantly worse outcomes in high transporters receiving APD [15]. Moreover, the few available outcome studies that have been published have been potentially limited by inadequate subgroup sample sizes and statistical power [1,10,15], study heterogeneity [5], vintage bias [3,15], centre effects [10,15], co-intervention bias (particularly icodextrin) [3,5] and lack of head-to-head comparisons of APD versus CAPD in high transporters [1,3,5,10,15]. Finally, high transporter status has also been associated with other conditions, such as an increased prevalence of inflammatory markers, malnutrition, hypoalbuminaemia and co-morbid illnesses [16–21], which may not be readily amenable to APD treatment.

The aim of the present study is to compare patient survival and death-censored technique survival in high transporters treated with APD versus those treated with CAPD, using data from the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry.

Subjects and methods

Study population

The study included all adult patients in Australia and New Zealand who started PD between 1 April 1999 and 31 March 2004. The data collected by the ANZDATA Registry consist of information on the underlying cause of end-stage renal disease (ESRD), demographic details, a limited range of co-morbidities (the presence of coronary artery disease, peripheral vascular disease, cerebrovascular disease, chronic lung disease, diabetes, hypertension and smoking), the type of each dialysis episode, details about kidney transplantation, and from 1 April 1999, measurements of height and dialysate plasma creatinine ratio at 4 hours (D–P Cr 4h). Peritoneal equilibration tests (PET) were requested to be performed within 6 months of commencement of PD, and at least 4 weeks apart from any peritonitis episodes.

For this study, APD was defined as the use of a cycler for PD treatment, including nightly intermittent peritoneal dialysis (NIPD), continuous cycling peritoneal dialysis (CCPD) or tidal peritoneal dialysis (TPD), nightly (NIPD) or continuous (CTPD). Peritoneal transport status was analysed as a categorical variable according to the four groupings of D–P Cr 4h values defined by Twardowski et al. (low, <0.50; low-average, 0.50–0.64; high-average, 0.65–0.80; and high, ≥0.81) [22]. Body mass index (BMI) was calculated from the quotient of the weight and the square of the height at the commencement of renal replacement therapy and was analysed as a categorical variable.

The outcomes examined were patient death and death-censored technique failure. If a patient died within 60 days after transfer to haemodialysis, then the death was attributed to PD, because such early deaths were considered to reflect the health status of patients during the period of failing PD therapy. In contrast, deaths that occurred in >60 days after cessation of PD due to renal transplantation were not attributed to PD, and such episodes were censored at the end of PD treatment. Death-censored technique failure was defined as a transfer from PD to haemodialysis for >1 month and was examined without counting death during treatment as a failure. Change of one PD modality to another was not considered as a technique failure. For both of these outcomes, survival time was calculated from the date of commencement of each PD episode to the date of death, transfer to haemodialysis, transplantation, loss of follow-up or 31 March 2004.

Statistical analysis

The patients with high transport membranes were classified into either CAPD or APD groups according to the PD modality at 90 days after PD commencement. Patients who completed <90 days on PD were excluded from the main analyses. Results were expressed as frequencies and percentages for categorical variables, mean ± standard deviation for continuous variables, and median and interquartile range for non-parametric data. Distributions of categorical variables across the two groups were compared by means of chi-square test and continuous variables by t-test if parametric and Mann–Whitney test if non-parametric.

Intention-to-treat analyses were performed for outcomes of patient mortality and death-censored technique failure using time-to-event analysis techniques. Univariable descriptions used Kaplan–Meier survival analyses. Multivariate Cox proportional hazards models were created using a backward stepwise elimination process based on the likelihood ratio test if P > 0.05. The other covariates included in the Cox models were gender, age, race, coronary artery disease, peripheral vascular disease, cerebrovascular disease, chronic lung disease, diabetes mellitus, smoking, BMI and peritoneal transport status. Standard errors were calculated using robust variance estimation for the correlated data, clustered according to the centre of initial treatment to address correlations within centres [23]. Proportional hazards assumptions were checked by Schoenfeld residuals and scaled Schoenfeld residuals, examined by formal hypothesis test and graphically. First-order interaction terms between the significant covariates were examined for all analyses. Statistical analyses were performed using Stata/SE 9.2 (College Station, TX, USA) statistical software.
Additional analyses

A separate as-treated analysis was performed by including PD modality as a time-dependent covariate. This analysis incorporated all patients, including those who completed <90 days on PD. A match-controlled analysis was also performed after controlling for age (categorical variable divided into four groups: <25, 25–50, 50–75 and >75 years), diabetes and racial origin.

Table 1. Baseline characteristics of the study populations

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>APD (n = 142)</th>
<th>CAPD (n = 486)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>54.5 ± 19.3</td>
<td>59.1 ± 15.6</td>
<td>0.003</td>
</tr>
<tr>
<td>Women</td>
<td>61 (43%)</td>
<td>205 (43%)</td>
<td>0.869</td>
</tr>
<tr>
<td>Racial origin</td>
<td></td>
<td></td>
<td>0.006</td>
</tr>
<tr>
<td>Caucasians</td>
<td>116 (82%)</td>
<td>320 (66%)</td>
<td></td>
</tr>
<tr>
<td>ATSI</td>
<td>8 (6%)</td>
<td>37 (8%)</td>
<td></td>
</tr>
<tr>
<td>MPI</td>
<td>11 (8%)</td>
<td>94 (19%)</td>
<td></td>
</tr>
<tr>
<td>Asians</td>
<td>5 (3%)</td>
<td>28 (6%)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>2 (1%)</td>
<td>7 (1%)</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>24.9 ± 5.3</td>
<td>25.6 ± 5.2</td>
<td>0.2</td>
</tr>
<tr>
<td>Underweight</td>
<td>22 (16%)</td>
<td>54 (11%)</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>59 (42%)</td>
<td>194 (40%)</td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
<td>38 (27%)</td>
<td>159 (33%)</td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>21 (15%)</td>
<td>79 (16%)</td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td>17 (12%)</td>
<td>79 (16%)</td>
<td>0.4</td>
</tr>
<tr>
<td>Chronic lung disease</td>
<td>18 (13%)</td>
<td>69 (14%)</td>
<td>0.6</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>53 (37%)</td>
<td>192 (40%)</td>
<td>0.6</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>34 (24%)</td>
<td>122 (25%)</td>
<td>0.8</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>24 (17%)</td>
<td>70 (14%)</td>
<td>0.6</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>44 (31%)</td>
<td>200 (41%)</td>
<td>0.03</td>
</tr>
<tr>
<td>D/P Cr 4h</td>
<td>0.88 ± 0.09</td>
<td>0.87 ± 0.07</td>
<td>0.2</td>
</tr>
<tr>
<td>Centre size</td>
<td></td>
<td></td>
<td>0.004</td>
</tr>
<tr>
<td>Large (≥101 patients per centre)</td>
<td>69 (49%)</td>
<td>316 (65%)</td>
<td></td>
</tr>
<tr>
<td>Moderately large (51–100 patients)</td>
<td>40 (28%)</td>
<td>99 (20%)</td>
<td></td>
</tr>
<tr>
<td>Moderately small (11–50 patients)</td>
<td>30 (21%)</td>
<td>62 (13%)</td>
<td></td>
</tr>
<tr>
<td>Small (≤10 patients)</td>
<td>3 (2%)</td>
<td>9 (2%)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: ATSI, Aboriginal and Torres Strait Islander; APD, automated peritoneal dialysis; BMI, body mass index; CAPD, continuous ambulatory peritoneal dialysis; D/P Cr 4h, dialysate-plasma creatinine ratio at 4 hours; MPI, Maori and Pacific Islander.

Additional analyses

A separate as-treated analysis was performed by including PD modality as a time-dependent covariate. This analysis incorporated all patients, including those who completed <90 days on PD. A match-controlled analysis was also performed after controlling for age (categorical variable divided into four groups: <25, 25–50, 50–75 and >75 years), diabetes and racial origin.

Results

Patient characteristics

Between 1 April 1999 and 31 March 2004, 4128 patients commenced PD in Australia and New Zealand. Six hundred and seventy-three (16%) of the total study population were high transporters. Forty-five patients with <90 days of follow-up on PD were excluded from the intention-to-treat analysis, such that the final analysis included 628 high transporters. There were 486 patients in the CAPD group and 142 in the APD group. The patients in the APD group were more likely to be younger than the CAPD group (54.5 ± 19.3 versus 59.1 ± 15.6 years, P = 0.003), Caucasian (82% versus 66%, P = 0.006) and less likely to be diabetic (31% versus 41%, P = 0.03) and treated in a large PD centre (49% versus 65%, P = 0.004) (Table 1). Mean baseline D–P Cr 4h in the APD group was comparable to the CAPD group (0.88 ± 0.09 versus 0.87 ± 0.07, P = 0.151).

Patient survival

One hundred and forty-eight patients died during the study period (24/142 or 17% on APD and 124/486 or 26% on CAPD). The patients in the APD group experienced a lower crude death rate [10.0 per 100 person-years, 95% confidence interval (CI) 6.7–14.9] than those in the CAPD group (14.6 per 100 person-years, 95% CI 12.2–17.3) (Figure 1). On univariate, Cox proportional hazards model analysis, the mortality rate of high transporters during APD treatment was significantly lower than that during CAPD treatment [unadjusted hazard ratio (UHR) 0.57, 95% CI 0.35–0.94] (Table 2 and Figure 1).

In the multivariate analysis, high transporters again experienced a significantly lower hazard of mortality during APD treatment compared to that during CAPD treatment [adjusted hazard ratio (AHR) 0.56, 95% CI 0.35–0.87]. Similar results were observed following additional adjustment for centre size (HR 0.56, 95% CI 0.35–0.90). The
other factors that were independently associated with a significantly altered hazard of mortality on APD versus CAPD in the high transporters were age (AHR 1.04, 95% CI 1.03–1.05), coronary artery disease (AHR 1.52, 95% CI 1.0–2.32) and BMI between 25 and 29.9 kg/m² (AHR 0.63, 95% CI 0.46–0.85). As compared to Caucasians, Aboriginal and Torres Strait Islanders had poorer survival (AHR 2.08, 95% CI 1.11–3.93), and Asians had better survival (AHR 0.43, 95% CI 0.21–0.88).

The hazard of death was comparable between APD and CAPD for low-average and high-average transporters, but patient survival was inferior in the APD group among low transporters (Table 2).

On as-treated analysis of 673 high transporters, patients receiving APD treatment (n = 310) experienced better survival than those receiving CAPD treatment (n = 363) on both univariate (UHR 0.74, 95% CI 0.56–0.97) and multivariate Cox proportional hazards model analyses (AHR 0.72, 95% CI 0.54–0.96).

A subgroup analysis was also performed to compare the outcomes of high transporters treated entirely with CAPD (n = 336) with those treated entirely with APD (n = 58). High transporters treated entirely with APD experienced better survival on univariate (UHR 0.33, 95% CI 0.16–0.67) and multivariate analyses (AHR 0.29, 95% CI 0.14–0.60). Finally, a matched case-control analysis was undertaken in which high transporters receiving APD or CAPD were matched for age, diabetes and racial origin. This analysis demonstrated a persistent survival benefit in the APD group (hazard ratio 0.60, 95% CI 0.36–0.96).

Death-censored technique failure
The crude death-censored technique failure rates of high transporters were similar between APD (21.3 per 100 person-years, 95% CI 16.2–28.0) and CAPD (20.5 per 100 person-years, 95% CI 17.7–23.8) (Figure 2). On univariate analysis (Table 3), high transporters had comparable death-censored technique failure during APD treatment to that during CAPD treatment (UHR 0.93, 95% CI 0.67–1.28).

On multivariate Cox proportional hazards model analysis, no significant differences were observed in death-censored technique survival of high transporters between APD and CAPD treatment (AHR 0.88, 95% CI 0.64–1.21) (Table 3). In the high transporters, none of the other

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**Table 2.** Results of intention-to-treat Cox proportional hazards model analyses of the relative hazard of APD versus CAPD for patient survival, according to peritoneal transport group

<table>
<thead>
<tr>
<th>Transport group</th>
<th>Univariate analysis</th>
<th>Multivariate analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR</td>
<td>95% CI</td>
</tr>
<tr>
<td>High (n = 628)</td>
<td>0.57</td>
<td>0.35–0.94</td>
</tr>
<tr>
<td>High-average (n = 1936)</td>
<td>0.98</td>
<td>0.72–1.34</td>
</tr>
<tr>
<td>Low-average (n = 1146)</td>
<td>0.70</td>
<td>0.46–1.07</td>
</tr>
<tr>
<td>Low (n = 196)</td>
<td>2.21</td>
<td>1.24–3.93</td>
</tr>
</tbody>
</table>

**Table 3.** Results of intention-to-treat Cox proportional hazards model analyses of the relative hazard of APD versus CAPD for death-censored technique survival, according to peritoneal transport group

<table>
<thead>
<tr>
<th>Transport group</th>
<th>Univariate analysis</th>
<th>Multivariate analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR</td>
<td>95% CI</td>
</tr>
<tr>
<td>High (n = 628)</td>
<td>0.93</td>
<td>0.67–1.28</td>
</tr>
<tr>
<td>High-average (n = 1936)</td>
<td>1.16</td>
<td>0.95–1.40</td>
</tr>
<tr>
<td>Low-average (n = 1146)</td>
<td>0.99</td>
<td>0.75–1.33</td>
</tr>
<tr>
<td>Low (n = 196)</td>
<td>1.35</td>
<td>0.79–2.28</td>
</tr>
</tbody>
</table>

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**Fig. 2.** Kaplan–Meier graph showing Death-Censored technique survival in 628 high transporters treated with APD or CAPD. The difference between the two groups was not statistically significant (log rank 0.17, P = 0.7).
variables included in the multivariate analysis were found to be independently associated with a significantly altered hazard of death-censored technique failure on APD versus CAPD. The hazard of technique failure was comparable between APD and CAPD for all other transport categories (low, low-average and high-average).

Discussion

The present study demonstrated that APD conferred a statistically significant and clinically important survival advantage in high transporters compared with CAPD. However, APD did not exert a beneficial effect on technique survival in high transporters and did not significantly influence either patient or technique survivals in high-average or low-average transporters. Our study is the first to have comprehensively examined the impact of APD versus CAPD in high transporters and supports the recommendations of the ISPD Ad Hoc Committee on Ultrafiltration Management in Peritoneal Dialysis [2].

The findings are in keeping with those of previous publications [1,3,5], which have suggested that the increased risk of death in high transporters is abrogated by APD. In a previous ANZDATA analysis of 3702 incident PD patients [1], high transporter status was found to be a significant, independent predictor of death-censored technique failure (HR 1.23, 95% CI 1.02–1.49) and mortality (HR 1.34, 95% CI 1.05–1.79) compared to low-average transport status. Subgroup analyses according to type of PD therapy demonstrated that high transporter status was independently predictive of mortality and death-censored technique failure for patients receiving CAPD, but not for those receiving APD. Similarly, a meta-analysis of 19 studies by Brimble et al. [5] estimated that high transporters had a 77% higher risk of mortality, after adjustment for age, diabetes and serum albumin concentration. This increased risk of death was inversely proportional to the percentage of patients who were treated with continuous cycling PD within any given study. Several other small single-centre studies have also reported no increased risk of death or technique failure in high transporters receiving APD [3,24].

In contrast, Hung et al. reported an increased risk of death in high transporters receiving APD compared with other transport groups. The apparent disparity in findings may potentially be explained by the fact that the investigation by Hung et al. involved an earlier vintage of PD patients (mid-1990s) and was a single-centre study.

The mechanisms underpinning an increased risk of death in high transporters have been variously attributed to inadequate ultrafiltration and fluid overload, malnutrition, increased dialysate protein losses and hypoalbuminaemia, and associated comorbid illnesses such as cardiovascular disease [16–21]. It has generally been proposed that the shorter dialysis dwells of APD therapy compared with CAPD would serve to minimize the negative impact of rapid glucose absorption by high transporters on peritoneal ultrafiltration, thereby mitigating fluid overload [2,4]. However, evaluating the impact of APD therapy on clinical outcomes in high transporters has often been confounded in most studies by the effects of co-intervention, particularly icodextrin administration [3]. Several studies have demonstrated that icodextrin induces a greater ultrafiltration response in high transporters than in other PD patients [25–27], and a randomized controlled trial has confirmed that icodextrin is associated with superior fluid removal in high transporters compared with conventional glucose dialysate solutions [25]. Although the precise extent of icodextrin prescription was unknown in the present study because the ANZDATA Registry does not collect such information, icodextrin was not widely available in Australia and New Zealand during the study period (1999–2004), such that the survival benefits observed in the present study are likely to predominantly reflect the effect of APD per se rather than a concomitant effect of icodextrin.

The propitious effect of APD on survival was strictly limited to the high transporter subgroup of PD patients. Outcomes between APD and CAPD were comparable for high-average and low-average transporters. Importantly, APD therapy was associated with inferior survival in low transporters. The mechanisms responsible for this novel finding are uncertain, although a previous study by Durand et al. [28] reported lower small solute clearances in low transporters treated with APD compared with CAPD due to diminished membrane–dialysate contact time as a result of multiple drain–fill sequences.

Interestingly, we found that APD did not exert any beneficial impact on death-censored technique failure in high transporters compared with CAPD. This observation may reflect the fact that inadequate ultrafiltration accounts for <10% of causes of technique failure in Australia and New Zealand [29], whilst the majority of cases are due to social reasons and infective complications, which may be less amenable to amelioration by APD. Alternatively, it is possible that any benefit conferred by APD on the technique survival of high transporters with impaired ultrafiltration is abrogated by a tendency of Australasian clinicians to promptly convert such patients to haemodialysis to try to improve their outcome, rather than persist with PD. Indeed, a previous ANZDATA study by our group has demonstrated that the heightened risk of death in high transporters on PD is abrogated following conversion to haemodialysis [30]. However, whether high transporters on PD are better off being treated with APD or transfer to haemodialysis has not been studied to date.

The strengths of this study lie in its large cohort size and the rigor and robustness of the statistical analyses performed. We included all patients receiving PD in Australia across 66 centres during the study period, such that a variety of centres were included with varying approaches to the treatment of peritonitis. This greatly enhanced the external validity of our findings. Time on each modality was accounted for by including PD modality as a time-dependent covariate. Moreover, as shown in other studies [31–34], the APD and CAPD groups exhibited significant differences with respect to a number of baseline characteristics; hence, all of these characteristics were adjusted for in the multivariate Cox proportional hazards models.

These strengths should be balanced against the study’s limitations. The observed associations between PD modality and patient outcomes according to baseline peritoneal transport status in this non-randomized observational study
do not establish causality. ANZDATA does not collect important information, such as patient compliance, individual unit management protocols (including peritoneal equilibration test protocols), PD prescription (dwell volume, exchange number, dwell time, drain and fill times, tidal versus non-tidal, dry periods, assisted or unassisted), ultrafiltration volumes, hydration status, use of icodextrin or other biocompatible PD fluids, laboratory values (such as C-reactive protein and serum albumin concentration), changes in peritoneal transport status over time or severity of comorbidities. Even though we adjusted for a large number of patient characteristics, the possibilities of indication bias and residual confounding could not be excluded. It was also not possible to elucidate the different pathophysiological mechanisms underpinning high transport status in our cohort [17,19,35]. Analysis of the relationships of PD modality with the different causes of technique failure (particularly peritonitis, ultrafiltration failure and inadequate small solute clearance) was not possible because of the way in which ANZDATA collects this information. The pattern and effect of loss of residual renal function in APD and CAPD patients on the various outcomes were also not able to be studied due to incomplete residual renal function data collection during the study period. Finally, we could not study the effect of the dialysis dose on the outcomes in CAPD and APD patients.

In conclusion, the present study demonstrated that APD treatment is associated with a significant overall survival advantage in high transporters compared with CAPD, although death-censored technique survival is comparable between the two modalities. On the other hand, APD treatment is associated with inferior survival in low transporters. In the absence of adequately powered randomized controlled trials of APD versus CAPD, the observed survival outcomes of this large registry study favour APD therapy for high transporters and CAPD therapy for low transporters.

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Conflict of interest statement. D.W.J. is a consultant for Baxter Healthcare Pty Ltd and has previously received research funds, travel funds and speakers’ honoraria from this company. He has also received speakers’ honoraria and research grants from Fresenius Medical Care. K.M.B. is a consultant for Baxter Healthcare Pty Ltd. S.P.M. has received speaking honoraria from Fresenius Australia and Baxter Australia. The results presented in this paper have not been published previously in whole or part, except in abstract format.

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