Home Versus Facility Dialysis and Mortality in Australia and New Zealand


Mortality remains one of the most important outcomes in dialysis, from the point of view of patients, caregivers, and health service sector stakeholders.1,4 Due to the lack of randomized controlled trials, the effect of dialysis modality on mortality has been uncertain. Up until the turn of the last century, observational studies suggested that peritoneal dialysis (PD) was associated with worse outcomes than hemodialysis (HD) in terms of mortality for the average patient, as well as uncertain benefits in terms health-related quality of life.4 More recent observational studies have suggested that PD has a reasonably similar survival to HD5-8 with better health-related quality of life.11-17 Consequently, modality choice (where choice is available) is often made largely on the basis of “hard” medical contraindications and lifestyle preference alone.18

We have previously analyzed mortality rates by modality in the Australia and New Zealand (ANZ) dialysis population. These early observational studies showed home HD to be associated with better survival than facility HD, and PD with worse survival.19-22 However, our subsequent observational studies have shown that mortality rates are improving faster for PD and home HD than for facility HD.23,24 In the current observational study, we compare mortality risk by dialysis modality in a cohort from 2013-2017, contrasting the findings with historical cohorts. We used data from the Australia and New Zealand Dialysis and Transplant Registry (ANZDATA), accounting for patient characteristics, treating center, and kidney transplantation as either a censoring or competing event.

We tested the hypothesis that all forms of home dialysis are associated with better survival than facility HD, and that peritoneal dialysis (PD) and dialysis in an unstaffed setting is a domiciliary or communal nature.25-28 Dialysis HD, mortality with CAPD and APD has improved over the years, with adjusted hazard ratios in 2013-2017 of 0.88 (95% CI, 0.78-0.99) and 0.91 (95% CI, 0.82-1.00), respectively. Increasingly, patients with lower clinical risk have been adopting APD, and to a lesser extent CAPD. Relative to facility HD, mortality with home HD was lower throughout the entire period of observation, despite increasing adoption by older patients and those with more comorbidities. All effects were generally insensitive to the modeling approach (initial vs time-varying modality, cause-specific versus subdistribution regression), different follow-up time intervals (5 year vs 7 year vs 10 year). There was no effect modification by diabetes, comorbidity, or sex.

Conclusions: The survival of patients on PD in 2013-2017 appears greater than the survival for patients on facility HD in ANZ. Additional research is needed to assess whether changing clinical risk profiles over time, varied dialysis prescription, and morbidity from dialysis access contribute to these findings.
was modeled as the modality at 90 days after dialysis inception in one set of models, and as varying modality throughout the period of observation in another set of models but with a 90-day lag in the attribution of death to a given modality. Both these modeling approaches have the effect of excluding patients who were not alive on dialysis at 90 days.

We justified this approach with the following 3 reasons. First, this sampling frame reduces contamination of the sample from acute kidney injury patients on dialysis who had been inadvertently entered into ANZDATA. Second, this sampling frame allows a reasonable time for patients to initiate training and transition onto home dialysis (~62% of those in ANZ who perform PD do so for the first time by 90 days, and about ~40% of corresponding home HD patients). Last, it is unlikely that modality has an impact upon early (ie, <90 days) mortality in incident dialysis patients; studies of early mortality identified other modifiable factors of more relevance to this often older and highly comorbid patient group.

The primary exposure of dialysis modality was defined to 2 ways. First, it was modeled as time varying, referring to a patient’s time-updated treatment modality over the entire study period. This is consistent with an as-treated approach (“did exposure that the patient initially received affect mortality?”). Second, it was modeled as fixed from 90 days, referring to a patient’s initial treatment modality at baseline. This is consistent with an intention-to-treat approach (“did exposure that the patient initially
received affect mortality, irrespective of subsequent changes that occurred along the way"). These are referred to as the “as-treated” (AT) and “intention-to-treat” (ITT) approaches, respectively. The primary outcome was patient death. We stratified analyses by era, defined by year of dialysis inception. In the main analysis, era was arbitrarily defined in 5-year windows (ie, 1998-2002, 2003-2007, 2008-2012, 2013-2017). Two other strategies were used as sensitivity analyses to address the well-known time-varying relative risk of mortality between PD and facility HD. One strategy used era defined in 7-year windows (1997-2003, 2004-2010, 2011-2017), and another used 10-year windows (1998-2007, 2008-2017). In all these different analyses, the hazard (or subhazard) ratio at time $T$ largely ignores the distribution of events before (and after) time $T$. The 5-year window can therefore be regarded as the best-case scenario for PD, with the mortality risk dominantly reflecting the early survival benefit for PD. By contrast, the 10-year window can be regarded as the worst-case scenario dominantly reflecting the late benefit for facility HD.

We adjusted estimates of the effect of modality on mortality using available patient-related risk factors: age, sex, ethnicity, primary kidney disease, estimated glomerular filtration rate (eGFR) at dialysis inception, late referral for nephrology predialysis care (<3 months before dialysis inception), diabetes mellitus (none, type 1, type 2), body mass index (BMI), medical comorbidity (coronary artery disease, peripheral vascular disease, cerebrovascular disease, chronic lung disease), and smoking.

Two modeling approaches were used. First, we used cause-specific proportional hazards models, censoring for kidney transplantation, return of kidney function, and loss to follow-up. We included a gamma-distributed shared frailty using the center of initial dialysis treatment as the random effect. Second, we used subdistribution proportional hazards (Fine and Gray) models, treating
transplantation as a competing risk. For all comparative analyses, facility HD was the reference category.

Two strategies were used for computations, as illustrated in Figure S1. These strategies all have different combinations of how modality was defined (AT vs ITT), what follow-up windows were used (5 years vs. 7 years vs. 10 years), and which type of models were implemented (cause-specific vs subdistribution). In the main analysis, we used AT dialysis modality with a 5-year follow-up window and a cause-specific proportional hazards model owing to our hypothesis comparing etiological risks.37-40 Of note, medical comorbidity was modeled as time-varying when the modality was similarly defined (AT), and modeled at 90 days when the modality was defined at baseline (ITT).

We used 3-way interaction terms in the main-effects model in a dataset containing patients from all eras, exploring potential effect modification by age, sex, diabetes mellitus, and presence of any comorbidity, based on plausible and previous findings from our group and others.41-47

Results

Cohort Description

The inception cohort for the main analysis contained 53,662 adult patients with 95,942 patient-years of follow-up. There were 52,097 people with 93,947 patient-years with 125,822 discrete periods of modality treatment without missing data: Table S1 summarizes the study

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<td>Age, y</td>
<td>60 [48-71]</td>
<td>63 [51-73]</td>
<td>63 [52-73]</td>
<td>63 [52-73]</td>
<td>&lt;0.001 (&lt;0.01)</td>
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<td>Diabetes mellitus</td>
<td>1,927 (35.8%)</td>
<td>3,329 (45.5%)</td>
<td>4,380 (51.8%)</td>
<td>4,790 (57.1%)</td>
<td>&lt;0.001 (0.15)</td>
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<tr>
<td>Comorbidity at baseline</td>
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<tr>
<td>Coronary artery</td>
<td>2,117 (39.4%)</td>
<td>3,167 (43.3%)</td>
<td>3,765 (44.5%)</td>
<td>3,213 (38.3%)</td>
<td>&lt;0.001 (0.05)</td>
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<tr>
<td>Peripheral vascular</td>
<td>1,429 (26.6%)</td>
<td>1,900 (26.0%)</td>
<td>2,387 (28.2%)</td>
<td>1,917 (22.8%)</td>
<td>&lt;0.001 (0.05)</td>
</tr>
<tr>
<td>Cerebrovascular</td>
<td>790 (14.7%)</td>
<td>1,149 (15.7%)</td>
<td>1,357 (16.1%)</td>
<td>1,107 (13.2%)</td>
<td>&lt;0.001 (0.03)</td>
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<td>Lung</td>
<td>837 (15.6%)</td>
<td>1,274 (17.4%)</td>
<td>1,621 (19.2%)</td>
<td>1,464 (17.5%)</td>
<td>&lt;0.001 (0.03)</td>
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<td>No. of patients</td>
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<td>2,885</td>
<td>2,461</td>
<td>2,368</td>
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<td>Age, y</td>
<td>62 [50-70]</td>
<td>63 [52-72]</td>
<td>63 [52-72]</td>
<td>63 [52-72]</td>
<td>&lt;0.001 (&lt;0.01)</td>
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<td>Diabetes mellitus</td>
<td>1,380 (40.3%)</td>
<td>1,263 (43.8%)</td>
<td>1,220 (49.6%)</td>
<td>1,256 (53.0%)</td>
<td>&lt;0.001 (0.10)</td>
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<td>Coronary artery</td>
<td>1,439 (42.1%)</td>
<td>1,112 (38.5%)</td>
<td>951 (38.6%)</td>
<td>784 (33.1%)</td>
<td>&lt;0.001 (0.07)</td>
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<td>Comorbidity at baseline</td>
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<tr>
<td>Peripheral vascular</td>
<td>1,007 (29.4%)</td>
<td>693 (24.0%)</td>
<td>599 (24.3%)</td>
<td>480 (20.3%)</td>
<td>&lt;0.001 (0.08)</td>
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<tr>
<td>Cerebrovascular</td>
<td>550 (16.1%)</td>
<td>414 (14.4%)</td>
<td>382 (15.5%)</td>
<td>287 (12.1%)</td>
<td>&lt;0.001 (0.04)</td>
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<tr>
<td>Lung</td>
<td>504 (14.7%)</td>
<td>445 (15.4%)</td>
<td>413 (16.8%)</td>
<td>319 (13.5%)</td>
<td>&lt;0.001 (0.03)</td>
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<td>1,097</td>
<td>1,555</td>
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<td>59 [47-70]</td>
<td>58 [46-69]</td>
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<tr>
<td>Diabetes mellitus</td>
<td>171 (39.6%)</td>
<td>446 (40.7%)</td>
<td>621 (39.9%)</td>
<td>1,044 (45.6%)</td>
<td>&lt;0.001 (0.05)</td>
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<tr>
<td>Coronary artery</td>
<td>177 (41.0%)</td>
<td>393 (35.8%)</td>
<td>497 (32.0%)</td>
<td>630 (27.5%)</td>
<td>&lt;0.001 (0.09)</td>
</tr>
<tr>
<td>Peripheral vascular</td>
<td>139 (32.2%)</td>
<td>239 (21.8%)</td>
<td>294 (18.9%)</td>
<td>365 (15.9%)</td>
<td>&lt;0.001 (0.11)</td>
</tr>
<tr>
<td>Cerebrovascular</td>
<td>75 (17.4%)</td>
<td>157 (14.3%)</td>
<td>194 (12.5%)</td>
<td>227 (9.9%)</td>
<td>&lt;0.001 (0.07)</td>
</tr>
<tr>
<td>Lung</td>
<td>64 (14.8%)</td>
<td>159 (14.5%)</td>
<td>226 (14.5%)</td>
<td>257 (11.2%)</td>
<td>&lt;0.001 (0.05)</td>
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<tr>
<td>No. of patients</td>
<td>275</td>
<td>229</td>
<td>299</td>
<td>433</td>
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<tr>
<td>Age, y</td>
<td>47 [38-54]</td>
<td>48 [41-56]</td>
<td>52 [44-59]</td>
<td>53 [45-61]</td>
<td>&lt;0.001 (0.04)</td>
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<tr>
<td>Diabetes mellitus</td>
<td>40 (14.6%)</td>
<td>39 (17.0%)</td>
<td>98 (32.8%)</td>
<td>187 (43.2%)</td>
<td>&lt;0.001 (0.27)</td>
</tr>
<tr>
<td>Coronary artery</td>
<td>43 (15.6%)</td>
<td>38 (16.6%)</td>
<td>57 (19.1%)</td>
<td>91 (21.0%)</td>
<td>0.27 (NA)</td>
</tr>
<tr>
<td>Peripheral vascular</td>
<td>25 (9.1%)</td>
<td>20 (8.7%)</td>
<td>27 (9.0%)</td>
<td>53 (12.2%)</td>
<td>0.35 (NA)</td>
</tr>
<tr>
<td>Cerebrovascular</td>
<td>21 (7.6%)</td>
<td>3 (1.3%)</td>
<td>15 (5.0%)</td>
<td>23 (5.3%)</td>
<td>0.005 (0.10)</td>
</tr>
<tr>
<td>Lung</td>
<td>17 (6.2%)</td>
<td>15 (6.6%)</td>
<td>27 (9.0%)</td>
<td>50 (11.6%)</td>
<td>0.04 (0.08)</td>
</tr>
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Continuous variables are shown as median [interquartile range]; categorical variables are shown as number (percentage). These descriptions are derived from the risk set for analyses of modality. Corresponding descriptions at the time of modality inception from the risk set for analyses of as-treated modality are provided in Table S3. Abbreviations: APD, automated peritoneal dialysis; CAPD, continuous ambulatory peritoneal dialysis; HD, hemodialysis.

*Effect size in parentheses. For effect size calculations, see Item S1. Effect size statistics describe the likely magnitude of differences; a value of 0.2 might be considered a small effect size, 0.5 a medium one, and 0.8 and greater a large effect size.
cohort and excluded cohort due to missing data at dialysis inception. The excluded cohort was a small proportion of the potential study cohort, with very small differences when compared with the study cohort.

Descriptive Data by Modality and Era

Table 1 summarizes the clinical characteristics of the study cohort by era of dialysis inception from the main analysis. Over the years, the key changes over time are (1) increases in type 2 diabetes mellitus and diabetic nephropathy, (2) decreases in recorded cardiovascular comorbidity at dialysis inception, (3) decreases in “late referrals” for dialysis, and (4) increases in overweight and obese patients.

Table 2 summarizes the clinical characteristics of the study cohort by ITT modality (ie, baseline modality at 90 days). The largest difference in modifiable risk factors between modalities can be found in the proportion of patients who are late referrals for dialysis, which is lowest with home HD, greatest with facility HD, and intermediate with PD. In addition, patients initially treated with home HD were younger compared with those treated with facility HD and PD, more likely to have kidney failure secondary to single-organ disease (eg, glomerulonephritis) rather than systemic disease, more likely to have a higher BMI, and less likely to have diabetes mellitus or medical comorbidity. Patients on CAPD and APD were generally similar to each other, and together were similar to those treated with facility HD. Table S2 replicates Table 2 but according to AT (ie, time-varying) modality, meaning that patients might be classified in multiple categories depending on the modalities that they were exposed to during the period of observation. The findings are generally similar to Table 2.

Table 3 summarizes key clinical characteristics of the study cohort at 90 days by by ITT modality (ie, baseline modality at 90 days) for each era. The following generalizations can be made. Over the years, there has been a small increase in age in the patients with an initial modality of facility HD or CAPD, no change in age in those with an initial modality of APD, but a comparatively large increase in age in those starting home HD. In all modalities, there has been an increase in the proportion of patients with diabetes mellitus, although this increase is approximately twice as large for home HD as it is for the other modalities. For facility HD, CAPD, and APD, there has been no change or a decrease in the proportion of patients with comorbidity. By contrast, for home HD there has been a directional increase in most types of comorbidity. Table S3 replicates Table 3 but according to AT (ie, time-varying) modality. The findings are generally similar to Table 3.

Deaths and Censoring Events

The unadjusted mortality rates for the cohort overall and by modality are illustrated from the main analysis in Figure 1. There has been an improvement in crude death rates in the cohort from ~15 deaths per 100 patient-years in 1998-2002 to ~11 in 2013-2017, although this improvement is limited to those treated with facility HD and CAPD and APD. The number of deaths (and causes of death) are provided in Table S4 for 5-year, 7-year, and 10-year follow-up windows.

Overall, CVD was the largest attributed cause of death across all eras, followed by withdrawal from dialysis, and then infectious mortality. Over the years, it is notable that there has been a slight decrease in cardiovascular mortality as a cause of death, but a proportionately larger decrease in infectious mortality. In terms of cause of death by modality, cardiovascular mortality accounts for a greater proportion of deaths on home HD compared with other modalities, infectious mortality a greater proportion of deaths on PD, and patient withdrawal a greater proportion on facility HD.

The number of censoring events (and causes of events) are also provided in Table S5 for the 5-year, 7-year, and 10-year follow-up windows. As expected, the transplantation rate was highest for those on home HD and was stable across era. It has been lowest for facility HD throughout, with a small decrease across era. It has been stable for CAPD, but rapidly rising for those on APD.

Main Results

Unadjusted nonparametric estimates of survival by era and modality from the main analysis are illustrated in
Figure S2. Qualitatively, there is generally a lower risk of death in more recent eras, with the exception of home HD where the risk of death in the most recent era is similar to that in previous ones. Quantitatively, as expected, there is significant modification of the effect of modality on mortality risk by era for the individually specified era terms ($P = 0.008$) and for the model overall ($P < 0.001$). Separate estimates were therefore computed for each era. There is subjectively good fit between the modeled and observed data for survival in these models for the main analysis (Fig S3), and no violation of the proportional hazards assumption for era ($P$ values all $\sim 0.2$) (Fig S4). As expected, there was time dependency for modality ($P < 0.001$ for CAPD and APD) (Fig S5), justifying analyses with different follow-up time windows.

The fully adjusted models from the main analysis (ie, for eras defined by 1998-2002, 2003-2007, 2008-2012, 2013-2017) are presented in Figure S6. For comprehensibility, a summary illustration is presented in Figure 2, showing the adjusted effects of modality on mortality risk, by era. The figure also shows corresponding effects from the other supplementary analyses that use cause-specific proportional hazards models with shared frailty but with different follow-up time windows.

The corresponding results from the supplementary analyses using subdistribution proportional hazards models are illustrated in Figure 3. There is no violation of the proportional subhazards assumption for era, as assessed by the regressor’s interaction with time in the full models ($P$ values all $\sim 0.4$). Figure 3 shows the adjusted effects of modality on mortality risk by era from this set of models for the different follow-up time windows.

The numerical values for estimates and 95% confidence intervals from all the illustrated effects are provided in Table S6. Overall, all models demonstrate consistent results: the mortality rate of facility HD, which was previously observed to be lower than CAPD and APD is now higher than CAPD and APD, and the mortality rate with home HD continues to be lower than all other modalities and reasonably stable. From the main analysis, mortality with CAPD and APD relative to facility HD have adjusted hazard ratios in 2013-2017 of 0.88 (95% CI, 0.78-0.99) and 0.91 (95% CI, 0.82-1.00), respectively. The corresponding estimate for home HD is 0.50 (95% CI, 0.40-0.64).
Other Results

There were no 3-way interactions between dialysis modality, era, and diabetes mellitus or comorbidity \((P = 0.5,\) and \(0.9,\) respectively) in a model derived from the main analysis but containing patients from all eras. There was a borderline interaction with sex \((P = 0.06)\) and a weak one with age \((P = 0.03)\). This effect modification is illustrated in Figure 4, which shows the fully adjusted effects of modality for 3 subgroups of age (18-54, 55-64, and \(\geq 65\) years), and for female and male patients. Broadly, the trends over era in each age group are directionally similar to the main analysis although more marked for older patients. The same applies for both female and male patients, although the trend is more marked for male patients.

Discussion

There are 3 major insights from this observational study. First, we show that PD is associated with a lower adjusted mortality risk than facility HD in the contemporary ANZ population, contrary to our previous studies that demonstrated the opposite.\(^{19,22,26}\) These findings are directionally similar to studies from Taiwan,\(^{48}\) the United States,\(^{44,49}\) Canada,\(^{50,51}\) Europe,\(^{52,53}\) and South Korea.\(^{54,55}\) Recent systematic reviews of mortality risk by modality report similar survival for HD and PD\(^{5,6}\) but synthesize mainly older data without formal testing for effect modification by era.

The reasons for the improved mortality risk with PD are not clear. One contribution could be from the marked decrease in PD peritonitis that has occurred within ANZ over the last decade (Fig S7). PD peritonitis will result directly in death in 2% to 5% of cases, and indirectly to several-fold more in the period immediately following apparent full recovery.\(^{56,57}\) Another consideration is increased selection bias over the years, with an increasing propensity for lower-risk patients to receive PD rather than HD. This situation is in contrast with other health jurisdictions that have reported corresponding secular trends in which it is apparent that increasingly higher risk patients are being treated with PD.\(^{53,55}\) In our study, patients on PD in 2013-2017 had a lower prevalence of diabetes mellitus when compared with facility HD, whereas for those in previous times it was comparable or even higher (Table 3). Mortality estimates in our models already include adjustments for this difference, as well as any other differences between groups arising for the variables in Table 2. The estimates are not adjusted for unmeasured variables, however, and it is likely that socioeconomic, medication-related, and health services factors are also more...
favorable to PD compared with facility HD. The observed secular improvement in mortality risk with PD might therefore reflect this residual confounding.

A final consideration for the differential improvement of mortality risk with PD is the increased icodextrin uptake in ANZ, which has increased from 0 to ~50% over the period of observation (Fig S8). This intervention probably decreases mortality risk in clinical trials.59-62

Although differences in survival according to subgroups were not among our a priori objectives, in the exploratory analyses we noted that the mortality rates of elderly and diabetic patients on PD were similar to those of HD patients. Once again, this finding must be regarded in the light of possible selection bias from imbalance of unmeasured confounders as described previously. Notwithstanding, our findings suggest that careful consideration of the role of PD in the elderly and those with diabetes mellitus may be needed and reinforce the paradigm of “patient-centered” shared modality decision making for all patients.

Finally, there has been a marked change in home HD epidemiology, with an increasing number of patients with diabetes and medical comorbidity using this modality. We cannot definitively pinpoint the reason for this expanded use of home HD, but it is known that this increase coincides with the wider adoption of “intensive hemodialysis” in ANZ (Figs S9 and S10). It is likely that this submodality of home HD is regarded by many practitioners in ANZ as being safe or even appropriate for patients at higher clinical risk.63 Despite this secular change in demographics and comorbidity, the adjusted mortality risk with home HD has not changed relative to facility HD. We cannot identify the exact reason for the stable mortality rates in the home HD population, but it is possible that the potential benefits of intensive HD may have contributed. Although this submodality has not been shown definitively to improve the mortality risk in clinical trials, it does improve left ventricular structure and function. In a previous analysis, we showed intensive dialysis in the home setting to be associated with lower mortality risk in ANZ.29

Our study is limited by the usual foibles of observational studies, such as ascertainment error in the recording of data, the potential for model misspecification, and residual confounding as noted previously. In addition, we assumed that patients did not change their dialysis center during treatment, although almost 9% of patients do change centers after day 90 of dialysis.

Figure 4. Hazard ratios (HRs) for death by era and modality the main model as highlighted in Figure 2, presented in different categories of age and gender (the marker represents point estimates, the whiskers 95% CI).
Finally, the clinical and organizational culture of dialysis delivery in ANZ is distinctive, wherein there is strong emphasis across all stakeholder groups on the primacy of home dialysis. As such, health care systems are well resourced for home dialysis, with health care workers who are well versed and confident with the modalities. In addition, there are evident practices in this study that are not customary elsewhere, such as the increasing trend to treat lower risk patients with PD rather than HD. The results observed in ANZ may therefore not be entirely generalizable elsewhere, although we note the directional similarities between our findings and those from other health jurisdictions.

In summary, we have identified that the survival for patients on PD now appears to be better than the survival for patients on facility HD. The relevance of this important finding will vary by health jurisdiction, and health care workers and funding agencies should be assessing the role of home dialysis in their own contemporary settings.

Supplementary Material

Supplementary File (PDF)

Figure S1: Analytical strategies.

Figure S2: Unadjusted nonparametric estimates of survival from the main analysis, by modality and era.

Figure S3: Kaplan-Meier observed survival curves by modality and era for the main analysis, compared with those predicted by cause-specific proportional hazards models with shared frailty.

Figure S4: Schoenfeld residuals plots from the main analysis showing no time dependency of effects for era.

Figure S5: Schoenfeld residuals plots from the main analysis showing time dependency of effects for CAPD and APD, justifying the sensitivity analyses with different follow-up time windows.

Figure S6: HRs for mortality by modality from the sensitivity analyses with different follow-up windows, era, and modality. Justifying Figure S5 showing no time dependency of effects for era.

Table 2, with shared frailty.

Figure S7: Peritonitis rates from Australian episodes collected in ANZDATA and from NZ episodes collected in the NZ PD registry.

Figure S8: Icodextrin point prevalence amongst PD patients at end of each year in ANZDATA.

Figure S9: Median frequency of dialysis in home HD patients at end of each year in the study cohort.

Figure S10: Median treatment session length of dialysis in home HD patients at end of each year in the study cohort.

Item S1: Detailed methods.

Table S1: Clinical characteristics of the inception cohort.

Table S2: Clinical characteristics of the study cohort at the time of modality inception, by as treated modality.

Table S3: Key clinical characteristics of the study cohort at the inception of each episode of modality, by modality and era.

Table S4: Number and causes of death in analyses, by follow-up window, era, and modality.

Table S5: Number and causes of censoring events in analyses, by follow-up window, era, and modality.

Table S6: Numerical estimates from the main and supplementary models as defined in Figure S1 and illustrated in Figures 2-4.

Article Information

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Home Versus Facility Dialysis and Mortality in Australia and New Zealand

**Setting & Participants**

52,097 adults starting dialysis in Australia and NZ 1998-2017

Mortality risk by modality compared between

- 1998-2002
- 2003-2007
- 2008-2012
- 2013-2017

Estimates computed using cause-specific and sub-distribution regression analysis

**Results**

Unadjusted Mortality Rates per 100 Patient-Years (Point Estimates, 95% CI)

Adjusted HR for mortality vs facility HD (Point Estimates, 95% CI)

Effects insensitive to competing risks, and not modified by diabetes, comorbidity, or sex.

Secular changes / differences were greatest for older patients.

**CONCLUSION:** Survival for patients treated with PD is greater than for those treated with facility HD in Australia and New Zealand.


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