Assessment of current practice and barriers to antimicrobial prophylaxis in peritoneal dialysis patients

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ABSTRACT

Background. Existing Australasian and international guidelines outline antibiotic and antifungal measures to prevent the development of treatment-related infection in peritoneal dialysis (PD) patients. Practice patterns and rates of PD-related infection vary widely across renal units in Australia and New Zealand and are known to vary significantly from guideline...
Adherence to evidence-based clinical practice guidelines is thought to improve patient outcomes, particularly when there are significant variations in practice, excessive morbidity and/or mortality is associated with a disease process, when treatment has the potential to reduce this, and when the services involved are costly [1]. Even though most healthcare workers are aware of the existence of evidence-based guidelines, many are unfamiliar with the content of these guidelines and they are not routinely adopted into clinical practice [2]. Active implementation programmes are needed to engage with stakeholders. It is also essential to identify areas of non-adherence to these guidelines and implement changes in clinical practice in accordance with guideline recommendations [3].

Background

Adherence to evidence-based clinical practice guidelines is thought to improve patient outcomes, particularly when there are significant variations in practice, excessive morbidity and/or mortality is associated with a disease process, when treatment has the potential to reduce this, and when the services involved are costly [1]. Even though most healthcare workers are aware of the existence of evidence-based guidelines, many are unfamiliar with the content of these guidelines and they are not routinely adopted into clinical practice [2]. Active implementation programmes are needed to engage with stakeholders. It is also essential to identify areas of non-adherence to these guidelines and implement changes in clinical practice in accordance with guideline recommendations [3].

Methods

Study design

The KHA-CARI Guidelines Peritoneal Dialysis Implementation Project is a prospective multicentre network study designed to assess the adherence to the KHA-CARI (published in 2004) [12] and ISPD guidelines (published in 2011) [13] targeting the prophylactic use of antibiotics at the time of Tenckhoff catheter insertion, the prophylactic use of antibiotics to prevent exit-site infection (ESI), and the prophylactic use of antifungals during courses of antibiotic therapy (Supplementary data, Appendix S1).

Results

Peritoneal dialysis is an accepted form of renal replacement therapy among Australians and New Zealanders with end-stage kidney disease (ESKD). More than 20% of all prevalent dialysis patients receive PD [4]. However, PD technique survival in Australia and New Zealand is lower than in many other parts of the world, attributed to a higher incidence of peritonitis [5, 6]. Clinical practice patterns are known to vary widely between centres in Australia and New Zealand and variations from clinical practice guidelines are known to contribute to poor infection outcomes [5]. In 2008, over 30% of Australian and New Zealand PD units did not meet the ISPD minimum accepted peritonitis rate of one episode per 18 patient-months (0.67 episodes per patient-year) [5, 7–10]. PD-related infections are also a key contributor to technique failure in PD patients in Australia and New Zealand with this being cited as the second and third most common cause of technique failure, respectively, in the 2012 ANZDATA registry report [9].

The Kidney Health Australia—Caring for Australasians with Renal Impairment (KHA-CARI) Guidelines are evidence-based clinical practice guidelines developed for use by healthcare workers primarily in Australia and New Zealand. The aim of these guidelines is to improve the health outcomes of patients with chronic kidney disease by adhering to best clinical practice and improving the quality and cost-effectiveness of the care provided. The International Society for Peritoneal Dialysis (ISPD) is an international organization dedicated to the dissemination of education and research in the area of PD. Both organizations have developed guideline recommendations for the prevention of infections in patients maintained on PD [11–13].

The KHA-CARI Guidelines PD Implementation Project commenced in 2011 with the task of trying to improve PD outcomes through adherence to these established PD guidelines. The aims of this study were to accurately measure each participating PD unit’s performance with regard to infectious outcomes and to identify the barriers and enablers to adherence to best practice guidelines in an attempt to close the gap of continuous quality improvement between evidence and outcomes.

This paper describes the baseline data obtained during the baseline phase for the subsequent guideline implementation project.
Participants

Eight PD units took part in the study. All Australian (78) and New Zealand (11) renal units were invited to participate by an 'Expression of Interest' letter that was sent out via the local professional nephrology society. Participating units were selected based on seven selection criteria defined by the steering committee (Supplementary data, Appendix S2).

Data collection

A project coordinator (D.C.) was responsible for managing the project, for conducting interviews on site, and collecting information on PD-related infections in incident PD patients on a case report form developed for the study. The interviews were semi-structured and aimed to identify key features of the clinical pathway that an incident PD patient with ESKD follows once referred to a nephrologist. A ‘process map’ was then developed for each PD unit.

Data were collected using quantitative and qualitative approaches. Quantitative data included demographic and PD-related infection data for incident patients who experienced a PD-related infection during a baseline monitoring period. Qualitative data were collected by the project coordinator through face-to-face interviews with staff at each participating unit. The nephrologist and PD nurse from each unit attended two face-to-face meetings, as did the members of the project’s steering committee. At the initial meeting, each PD unit was asked to outline what they thought were the barriers and enablers to optimal infection prevention at their unit. The units were given a cause-and-effect diagram to help identify barriers and enablers to good clinical practice [14]. Data on each PD unit’s protocols, policies and usual practices were also collected by asking each unit to complete a survey.

Information for each unit on PD-related peritonitis was obtained from the ANZDATA registry for the 12 months from 1 January 2011 to 31 December 2011. The units were asked to provide data on ESI for the same period. Currently, data on ESI are not submitted to ANZDATA.

Study outcomes

The following outcomes were assessed: (i) identification of key elements of current practice; (ii) rates of ESI and PD-related peritonitis and (iii) identification of the barriers and enablers relating to the guideline recommendations for the prevention of catheter surgery-related infection, ESI/tunnel infection and PD-related peritonitis.

STATISTICAL ANALYSIS

Results are expressed as frequencies and percentages for categorical variables, and medians and interquartile ranges for continuous variables. Peritonitis and ESI rates were calculated by totalling all the peritonitis or ESI episodes that occurred during the entire time on PD for all patients at each unit in the programme during the period 1 January 2011 to 31 December 2011. The total was then divided by the time at risk in years.

This follows the suggested method for reporting outlined by the ISPD [13].

Data were analysed using the software package SAS version 9.3 (SAS Institute, Cary, NC, USA).

RESULTS

Unit and patient characteristics

Of the 10 PD units that responded, 8 were selected by the steering committee. These units treated a total of 582 PD patients, which comprise 28.1% of the total PD population in Australia and New Zealand [9]. The numbers of patients receiving care in the individual units during the period 1 January 2011 to 31 December 2011 were divided into quartiles and centre size was categorized accordingly. Two of the units were categorized as large (>107 patients), two were categorized as medium-large (57–107 patients), two were classified as small-medium (32–56 patients) and two units were classified as small (<32 patients). There was one Australian PD unit with an inner regional location; the rest were located in major cities. The two PD units that were not selected were based at large metropolitan hospitals. All patients in all units had access to the full range of renal replacement therapy as all eight units offered both haemodialysis and PD. Four of the eight units were also transplanting centres and the models of care in Australia and New Zealand are hub and spoke so that all patients at all centres had access to transplantation as well. PD is a home-based therapy in Australia and New Zealand. All patients trained would be expected to be self-sufficient in the technique either alone or with the assistance of their spouse/carer.

Table 1 shows the baseline demographic characteristics of patients in the individual units and the overall PD cohort. There were no significant differences between the PD units for sex, age, body mass index or diabetes mellitus. There were significant differences between PD units for race, number of patients per unit, duration per patient of treatment with PD and the numbers receiving ambulatory peritoneal dialysis (APD) or continuous ambulatory peritoneal dialysis (CAPD). The number of patients on APD and CAPD ranged from 26 to 89% and 11 to 74%, respectively.

Current practice and organizational features

There was considerable variation in nurse to patient ratios (median: 1:19, range: 1:12–1:58) and the proportion of experienced surgeons to junior surgeons working at a unit (median: 2:05, range: 0:1–5:2). The NZ unit did not have an experienced surgeon available to it for Tenckhoff catheter insertions. A majority of the PD units completed patient PD training within 1 week (1–2 weeks) and seven of eight units offered home visits after commencement of PD.

Prophylactic antibiotics

All units had protocols in place for the administration of intravenous antibiotics to patients prior to catheter surgery. Seven of eight units had protocols for the routine prescription of antibiotic prophylaxis against ESI and five of eight units had
Table 1. Demographic characteristics and dialysis details of PD patients at the eight participating units

<table>
<thead>
<tr>
<th>Variables</th>
<th>Unit 1</th>
<th>Unit 2</th>
<th>Unit 3</th>
<th>Unit 4</th>
<th>Unit 5</th>
<th>Unit 6</th>
<th>Unit 7</th>
<th>Unit 8</th>
<th>P-value All Units</th>
<th>Australian PD population</th>
<th>New Zealand PD population</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. (%) patients</td>
<td>120 (21)</td>
<td>67 (12)</td>
<td>34 (6)</td>
<td>181 (32)</td>
<td>21 (4)</td>
<td>95 (17)</td>
<td>18 (3)</td>
<td>46 (8)</td>
<td>582 (100)</td>
<td>2074</td>
<td>792</td>
</tr>
<tr>
<td>No. (%) males</td>
<td>66 (55)</td>
<td>34 (51)</td>
<td>15 (44)</td>
<td>101 (56)</td>
<td>12 (57)</td>
<td>61 (64)</td>
<td>9 (50)</td>
<td>24 (52)</td>
<td>0.58</td>
<td>322 (55)</td>
<td>1164 (56)</td>
</tr>
<tr>
<td>No. (%) Caucasian</td>
<td>93 (78)</td>
<td>47 (70)</td>
<td>12 (35)</td>
<td>98 (54)</td>
<td>39 (57)</td>
<td>43 (45)</td>
<td>16 (89)</td>
<td>12 (26)</td>
<td>&lt;0.001</td>
<td>371 (64)</td>
<td>1482 (72)</td>
</tr>
<tr>
<td>No. (%) Aboriginal/Torres Strait Islander</td>
<td>6 (5)</td>
<td>1 (1)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0.006</td>
<td>365 (66)</td>
<td>0.54</td>
</tr>
<tr>
<td>No. (%) Maori/Pacific Islander</td>
<td>6 (5)</td>
<td>5 (8)</td>
<td>0 (0)</td>
<td>17 (9)</td>
<td>0 (0)</td>
<td>3 (3)</td>
<td>0 (0)</td>
<td>19 (41)</td>
<td>&lt;0.001</td>
<td>92 (16)</td>
<td>0.001</td>
</tr>
<tr>
<td>No. (%) Asian</td>
<td>11 (9)</td>
<td>5 (8)</td>
<td>0 (0)</td>
<td>58 (32)</td>
<td>8 (38)</td>
<td>25 (26)</td>
<td>2 (11)</td>
<td>14 (30)</td>
<td>&lt;0.001</td>
<td>400 (70)</td>
<td>0.001</td>
</tr>
<tr>
<td>No. (%) Other/unknown</td>
<td>4 (3)</td>
<td>4 (6)</td>
<td>0 (0)</td>
<td>7 (4)</td>
<td>1 (5)</td>
<td>7 (7)</td>
<td>0 (0)</td>
<td>1 (2)</td>
<td>0.52</td>
<td>23 (4)</td>
<td>40 (2)</td>
</tr>
<tr>
<td>Age in years (median, IQR)</td>
<td>2 (median, IQR)</td>
<td>27 (9)</td>
<td>26 (8)</td>
<td>27 (7)</td>
<td>26 (7)</td>
<td>26 (7)</td>
<td>25 (7)</td>
<td>25 (7)</td>
<td>0.10</td>
<td>26 (7)</td>
<td>26 (7)</td>
</tr>
<tr>
<td>Duration of dialysis in years (PD), median (IQR)</td>
<td>1.8 (3.2)</td>
<td>1.9 (3.4)</td>
<td>1.3 (1.9)</td>
<td>1.7 (3.6)</td>
<td>1.9 (1.6)</td>
<td>2.4 (3.4)</td>
<td>3.4 (5.2)</td>
<td>2.4 (2.9)</td>
<td>&lt;0.001</td>
<td>2.1 (2.7)</td>
<td>0.57</td>
</tr>
<tr>
<td>ESI rate (unit per patient-year)</td>
<td>0.06 (0.53)</td>
<td>0.13 (0.08)</td>
<td>0.03 (0.03)</td>
<td>0.41 (0.86)</td>
<td>0.31 (0.64)</td>
<td>0.53 (0.79)</td>
<td>0.82 (1.39)</td>
<td>0.36 (0.83)</td>
<td>&lt;0.001</td>
<td>0.14 (0.27)</td>
<td>0.14 (0.27)</td>
</tr>
</tbody>
</table>

*The χ² test was used for univariate analysis among units. Units represented: Princess Alexandra Hospital, QLD, Australia; Royal Brisbane and Women’s Hospital, QLD, Australia; Gosford Hospital, NSW, Australia; Regional Renal Dialysis Centre, Blacktown, NSW, Australia; Home Therapies Unit, Western Hospital, VIC, Australia; Monash Health, Adult, VIC, Australia; Home Therapies Service, Royal Hobart Hospital, TAS, Australia; Auckland City Hospital, New Zealand.

PD-related infection definitions

We found that the definitions of the PD-related infectious outcomes were not consistent across the units. Three units were using peritonitis definitions that differed from the ISPD definition [8]. Definitions of ESI and tunnel infection used differed from ISPD definitions in five and six units, respectively (Table 2).

Current ESI and peritonitis rates

Figure 1 shows that the reported peritonitis rates for the selected units for the period 1 January 2011 to 31 December 2011, ranged from 0.31 to 0.86 (95% CI: 0.23–1.39) episodes per patient-year. The reported ESI rates varied from 0.06–0.53 (95% CI: 0.03–0.83) episodes per patient-year (Figure 2). The two large and the two medium-large Australian units had the best peritonitis rates and three of these units also had the best ESI rates. One of the small-medium size units and one of the small units had the worst peritonitis rates; the same two units and the other small size unit had the worst ESI rates.

Of the six units that had a protocol prescribing ESI antibiotic prophylaxis, those that applied an antibiotic to the exit site had better ESI rates than those that used nasal application. Of the two units that did not have such a protocol, one had a good ESI rate (Unit 4) while one had a poor ESI rate (Unit 3) [Table 3]. Of the five units that had a protocol directing that an antifungal be given with any antibiotic course, two units had peritonitis rates close to the ISPD recommended standard of 0.36 episodes per patient-year. However, three units had peritonitis rates of 0.42 episodes per patient-year or greater. Interestingly, of the two units without this protocol, one unit had a good peritonitis rate while the other had a poor one (Table 3). Thus, from the data there was not an obvious correlation between the presence of protocols and the perceived application of the protocols to the rates of infection.

Perceived barriers to prophylaxis against PD-related infections

Perceived organizational, medical staff and patient barriers to the use of antibiotic prophylaxis are summarized in Figure 3. At the time of Tenckhoff catheter insertion, the most common barrier reported by medical staff was the nominated person forgetting to give an antibiotic at surgery whereas the most common organization-based barrier was the lack of a centralized patient database so that staff could not check whether or not the antibiotic had been administered. Fifty percent of the units did not have a centralized patient database.

The most common barriers to ESI prophylaxis reported by medical staff was concern by infectious diseases staff that routine use of mupirocin would result in the development of antibiotic resistance, difficulty in gaining agreement on the routine use of mupirocin at the exit site at units with more nephrologists on staff, and insufficient staff to test and treat patients if nasal carriage of *Staphylococcus aureus* was to be routinely monitored. The most common organization-based barrier was when the unit had a blanket policy against the routine use of protocols for the routine prescription of an antifungal agent during courses of antibiotics.
<table>
<thead>
<tr>
<th>Criteria used to define an ESI</th>
<th>ISPD definition</th>
<th>Unit 1 definition</th>
<th>Unit 2 definition</th>
<th>Unit 3 definition</th>
<th>Unit 4 definition</th>
<th>Unit 5 definition</th>
<th>Unit 6 definition</th>
<th>Unit 7 definition</th>
<th>Unit 8 definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of purulent drainage, with or without erythema of the skin at the catheter-epidermal interface</td>
<td>Presence of purulent drainage, with or without erythema of the skin at the catheter-epidermal interface</td>
<td>Presence of purulent drainage and erythema around the exit site</td>
<td>Presence of purulent exudate and/or pain and/or redness</td>
<td>Presence of the following in various combinations: pain/tenderness; bright pink or red colour &gt;13 mm; crust present; scab present; drainage – purulent or bloody; drainage—spontaneous or after pressure on sinus; wet exudate on dressing; swelling present; slightly exuberant or ‘proud flesh’</td>
<td>Presence of purulent discharge; or redness with non-purulent discharge and swab of exit site shows significant growth</td>
<td>Presence of purulent discharge, with or without redness</td>
<td>Presence of purulent discharge and/or significant polymorphonuclear cells from wound swab Gram stain, with or without positive culture</td>
<td>Presence of redness &gt;3 mm with purulent discharge</td>
<td>Presence of redness &gt;3 mm with purulent discharge</td>
</tr>
</tbody>
</table>

| Criteria used to define a tunnel infection | Presence of erythema, oedema or tenderness over the subcutaneous pathway | Presence of purulent drainage, oedema, erythema and tenderness around the exit site and along the position of the tunnel | Presence of pain/tenderness and/or swelling along the tunnel, with or without exit site infection; ultrasound of the tunnel identifying fluid collection | Presence of redness (subcutaneous tunnel) and/or tenderness with an ESI or peritonitis | Presence of redness, swelling at the tunnel with discharge at the exit site | Presence of redness, swelling at the tunnel with discharge at the exit site |

| Criteria used to define peritonitis | Presence of cloudy effluent with ≥100 white blood cells/µL with ≥50% polymorphonuclear cells; or abdominal pain; or positive microbiological culture of dialysate fluid | Presence of PD effluent with ≥100 white blood cells/µL with ≥50% polymorphonuclear cells; or cloudy effluent; or fever; or abdominal pain; or patient is systemically unwell | Presence of cloudy effluent with ≥100 white blood cells/µL with ≥50% polymorphonuclear cells; or cloudy effluent | Presence of cloudy effluent (≥100 white blood cells/µL) with ≥50% polymorphonuclear cells; or cloudy effluent with ≥100 white blood cells/µL with ≥50% polymorphonuclear cells and positive Gram stain; or abdominal pain | Presence of cloudy effluent with ≥100 white blood cells/µL with ≥50% polymorphonuclear cells and/or symptoms of peritoneal inflammation (fever, abdominal pain, vomiting, chills, nausea, abdominal tenderness); and/or recent identification of organism on Gram stain or culture of PD effluent | Presence of effluent with >100 white blood cells/µL with ≥50% polymorphonuclear cells; and/or abdominal pain; and/or symptoms of peritoneal inflammation (fever, abdominal pain, vomiting, chills, nausea, abdominal tenderness); and/or recent identification of organism on Gram stain or culture of PD effluent | Presence of effluent with >100 white blood cells/µL with ≥50% polymorphonuclear cells; and/or abdominal pain; and/or symptoms of peritoneal inflammation (fever, abdominal pain, vomiting, chills, nausea, abdominal tenderness); and/or recent identification of organism on Gram stain or culture of PD effluent | Presence of effluent with >100 white blood cells/µL with ≥50% polymorphonuclear cells; and/or abdominal pain; and/or symptoms of peritoneal inflammation (fever, abdominal pain, vomiting, chills, nausea, abdominal tenderness); and/or recent identification of organism on Gram stain or culture of PD effluent |
antibiotics at the exit site. Of the various patient-based barriers identified, the most common was distance from the PD unit and when the patient’s first language was other than English. Perceived barriers to appropriate antifungal prophylaxis are outlined in Figure 4. The most common medical staff-based barriers included lack of awareness of junior doctors or of doctors outside the hospital system of the need to co-prescribe antifungal medication with an antibiotic course. The only organization-based barrier identified was the lack of a formal policy around the need for antifungal medication when an antibiotic course is prescribed to a PD patient. Of the patient-based barriers identified, the most common barriers were the taste of the medication, the patient’s lack of understanding of the importance of taking the medication, and the cost of the medication. Only six fungal infections were reported over the study period, with four of them occurring in units where antifungal prophylaxis was not routine.

DISCUSSION

We found wide variation in the current practices in place at each PD unit. We found that the clinical staff were generally aware of these clinical practice guideline recommendations but there was considerable variation in application of the recommendations in practice. All units had protocols in place for two of the three guideline recommendations of interest to this project but the variability in peritonitis and ESI rates experienced by the units raised questions as to how well these processes were being followed.

We also identified a wide variation in the PD-related infection rates at the units. The peritonitis rates for the Australian

Table 3. Comparison of the exit site and peritonitis rates with unit protocols (January to December 2011)

<table>
<thead>
<tr>
<th>Unit number</th>
<th>Protocol to administer antibiotic prophylaxis at catheter surgerya</th>
<th>Protocol to prescribe ESI antibiotic prophylaxis</th>
<th>Protocol to prescribe antifungal with antibiotic course</th>
<th>ESI rate (episodes/patient-year)</th>
<th>Peritonitis rate (episodes/patient-year)</th>
<th>Fungal peritonitis rate (episodes/patient-year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unit 1</td>
<td>Yes (IV cephazolin intra-operatively)a</td>
<td>Yes—nasalb</td>
<td>Yes</td>
<td>0.26</td>
<td>0.39</td>
<td>0.00</td>
</tr>
<tr>
<td>Unit 2</td>
<td>Yes (IV cephazolin intra-operatively)a</td>
<td>Yes—exit site</td>
<td>Yes</td>
<td>0.13</td>
<td>0.40</td>
<td>0.08</td>
</tr>
<tr>
<td>Unit 3</td>
<td>Yes (IV cephazolin intra-operatively)a</td>
<td>Yes—nasalb</td>
<td>No</td>
<td>0.53</td>
<td>0.78</td>
<td>0.00</td>
</tr>
<tr>
<td>Unit 4</td>
<td>Yes (IV cephazolin intra-operatively)a</td>
<td>No (policy against routine use of mupirocin)</td>
<td>No</td>
<td>0.06</td>
<td>0.31</td>
<td>0.005</td>
</tr>
<tr>
<td>Unit 5</td>
<td>Yes (IV cephazolin intra-operatively)a</td>
<td>Yes—exit site</td>
<td>Yes</td>
<td>0.21</td>
<td>0.46</td>
<td>0.00</td>
</tr>
<tr>
<td>Unit 6</td>
<td>Yes (IV cephazolin pre- or intra-operatively)a</td>
<td>Yes—exit site</td>
<td>Yes</td>
<td>0.11</td>
<td>0.42</td>
<td>0.01</td>
</tr>
<tr>
<td>Unit 7</td>
<td>Yes (IV cephazolin intra-operatively)a</td>
<td>Yes—nasalb</td>
<td>No</td>
<td>0.27</td>
<td>0.86</td>
<td>0.00</td>
</tr>
<tr>
<td>Unit 8</td>
<td>Yes (IV cefuroxime on induction, 8 h post, 16 h post)a</td>
<td>Yes—nasalb</td>
<td>No</td>
<td>0.23</td>
<td>0.52</td>
<td>0.04</td>
</tr>
</tbody>
</table>

aVancomycin given to MRSA-positive patients and those allergic to cephazolin or cefuroxime.
bPatients screened every 6 months for S. aureus nasal carriage and treated with mupirocin ointment if found positive.
Only patients who screened positive for S. aureus nasal carriage at initial screening are screened every 6 months and treated with mupirocin ointment if found positive.
Only patients who screened positive for S. aureus nasal carriage at initial screening are screened every 3 months and treated with mupirocin ointment if found positive. The treatment is repeated for the following 2 months. Those that are negative are re-swabbed annually.
units ranged from 0.31 to 0.86 episodes per patient-year while the New Zealand unit had a rate of 0.52 episodes per patient-year. These compare with median rates for all PD units in Australia in 2011 of 0.55 episodes per patient-year and a median rate of 0.62 episodes per patient-year for all New Zealand PD units [4]. Most of the units in this study did not meet the ISPD minimum recommended standard of 0.36 episodes per patient-year and the rate was vastly inferior to the reported rate for countries such as Korea, Japan, Hong Kong and China [5]. The ESI rates for the study units varied hugely from 0.06 to 0.53 episodes per patient-year. It is not possible to compare these rates with other PD units in Australia and New Zealand because this information is not collected by the ANZDATA Registry. Other countries have reported ESI rates of 0.16–0.40 episodes per patient-year [15–18].

Several factors were identified that may explain the high rates of ESI and peritonitis seen at some units. Some of the selected PD units failed to adhere to the recommendations of the KHA-CARI and ISPD guidelines, particularly those on the routine use of antibiotics to prevent ESI and the need to co-prescribe antifungal medication when any course of antibiotics was given to a PD patient. Possible reasons for the non-adherence to guideline recommendations include the lack of policies/protocols (three units), awareness of and attitudes towards the guideline recommendations (two units), low (suboptimal) staff to patient ratios (two units), lack of access to an

**FIGURE 3:** Perceived barriers to appropriate antibiotic prophylaxis in PD patients.

**FIGURE 4:** Perceived barriers to appropriate antifungal prophylaxis in PD patients.
experienced surgeon to do the catheter placement (one unit) and resistance by infectious diseases staff to the routine use of mupirocin (three units). No units identified the quality of the evidence underpinning the KHA-CARI guidelines or the ISPD guidelines as a reason for non-adherence.

The fact that some units had protocols in place consistent with clinical guideline recommendations and yet appeared to have had less favourable outcomes in terms of infection rates and some units appeared to have better outcomes but no formal protocols suggests that other factors/variables are likely to explain these results (e.g., patient selection, definition of infection). In addition, although protocols consistent with the published clinical trial data were in place, the application or implementation of the protocols might have been suboptimal. Furthermore, patients entered into clinical trials are selected on particular criteria and may not be totally representative of the broad population treated in a study such as this one.

The perceived barriers to putting into practice the guideline recommendations show the range of barriers that can occur across different levels of healthcare. The perceived barriers were identified at three levels and included awareness of the relevant guideline recommendation and attitude to the guideline (healthcare providers); practical and cost impediments, knowledge and compliance issues (patients); and care processes, staffing and capacities (organization).

The clinical uptake or implementation of guideline recommendations does not automatically follow the production and dissemination of clinical practice guidelines. Recommendations are not always put into practice and many patients do not receive evidence-based care [2, 19, 20]. Research into guideline implementation has found that strategies such as interactive small group meetings, educational outreach visits, reminders, computerized decision support, introduction of computers in practice, substitution of tasks, multiprofessional collaboration, mass media campaigns and financial interventions are effective at changing practice [21]. The use of educational materials, conferences/courses, opinion leaders, education, performance feedback and patient-mediated interventions were found to yield mixed effects in terms of practice change. Total quality management/continuous quality improvement strategies were found to have only limited effects. Most of the interventions studied had some effects on care improvement with an average change of ~10% for main targets [21].

This study has a number of strengths. First, current practice data and individual patient data on ESI for the year prior to the start of the study were directly collected from the units. This gave an accurate assessment of the pathways from catheter insertion to the development of infection. Secondly, the combined patients at the eight selected PD units treated 28.1% of the total population of PD patients in Australia and New Zealand and therefore provided a good evaluation of PD practices in our region. The selected PD units were of varying sizes and locations and were identified as a good representation of the variability seen in the two countries. These features may make the results potentially generalizable to other PD patient populations. Barriers to the successful implementation of the antibiotic and antifungal prophylaxis guideline recommendations were identified by mapping the steps in the care process and by using the National Institute of Clinical Studies barrier tool [22].

This study also has some limitations. Due to resource constraints, more units were not able to be enrolled in the study. Second, the fact that the three process steps of interest were not directly audited was a limitation. For example, while the participating units were asked if they gave suitable antibiotics at catheter insertion, each unit was not specifically audited to establish if the antibiotics were reliably recorded when given and if they were given within an appropriate timeframe. In addition, patient-based barriers were reported by the healthcare professionals and not obtained directly from patient interviews. Furthermore, six of the seven Australian PD units have a ‘major city’ classification according to the Australian Bureau of Statistics remote area index, which makes the patient population sample a mostly urban one [23]. Finally, participation in the study was voluntary with only 10 units offering to join the study.

Implications for clinical practice

The active implementation of nephrology guidelines based on appropriate evidence for reducing infections has the potential to improve a unit’s PD-associated infection rates, catheter loss and technique failure rates, as a consequence. Although there are some caveats around the quality of evidence in some areas of PD practice, the consistent use of appropriate antibiotics at catheter insertion should be associated with reduced occurrence of post-surgery ESI and peritonitis. Our study does not conclusively show that mupirocin prevents exit-site/tunnel infection and peritonitis. Although antifungal prophylaxis can be beneficial, its use by individual PD units should depend on the background rate of fungal peritonitis and local geographical and patient demographic factors, as demonstrated in our study, whereby one of the units that had such a protocol had a higher fungal peritonitis rate than the three units that did not have an antifungal protocol in place.

Implications for clinical research

Further research is needed to determine which prophylactic strategies involving the application of antibacterial creams or solutions are most effective in preventing catheter ESI. Our results suggest that the units that used nasal mupirocin prophylaxis generally had higher ESI and peritonitis rates than those that used exit-site mupirocin prophylaxis. It has previously been noted that there have been no direct head-to-head comparison studies of intranasal mupirocin against either exit-site mupirocin or exit-site gentamicin [6]. In addition, the unit with the best infection rates did not use antibiotics at all but used daily application of povidone-iodine at the exit site.

As the barriers to adherence to clinical practice guidelines vary from individual to individual and from unit to unit, the results of a barrier analysis in one setting may not be generalizable to another. Research into the barriers to guideline adherence at a unit will benefit from being conducted locally and any implementation plan to improve adherence will need to address the identified factors.

In conclusion, this study has identified several factors that may contribute to the high rates of ESI and peritonitis that
occur in Australia and New Zealand compared with accepted international standards. Nearly all of these factors are potentially modifiable through improved education, creation of a centralized patient database and routine checking of patient exit-site care and exchange technique. The findings of this study will provide the first step in improving PD outcomes by having identified a number of perceived barriers and enablers to good antibiotic and antifungal practice in PD patients. Having an understanding of the barriers to good clinical practice that exist within each organization can inform the development of a targeted implementation strategy aimed at improving PD-related infection outcomes [3].

**SUPPLEMENTARY DATA**

Supplementary data are available online at http://ndt.oxfordjournals.org.

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**CONFLICT OF INTEREST STATEMENT**

F.G.B. is a consultant for Baxter Healthcare and Fresenius and has received travel grants from Amgen and Roche. M.P.G. has received honoraria from Amgen and Roche in the past 3 years for speaking at educational meetings. D.W.J. has received consultancy fees, speakers’ honoraria, travel sponsorships and research funding from Fresenius Medical Care and Baxter Healthcare. S.K.K. has received conference or meeting sponsorships from Shire Australia P/L, Roche, Amgen and Novartis and has received speaker honoraria from Merck, Sharp and Dohme. W.H.L. is on the advisory boards of Novartis and Alexion and has received research funds from Novartis and Pfizer. W.S. has received speakers’ honoraria from Baxter Healthcare. K.S. has received speaker’s honoraria from Baxter Healthcare and Boehringer Ingelheim and conference or meeting sponsorships from Shire Australia P/L, Roche, Boehringer Ingelheim, Amgen and Novartis. NDT has received consultancy fees, speakers’ honoraria and travel sponsorships from Amgen and Shire, and research funding from Amgen, Shire and Baxter Healthcare. R.G.W. has served on Medical Advisory Boards for Baxter and Fresenius and has received research funds and travel grants from Amgen, Roche and Janssen-Cilag. D.W.M. has received consultancy fees, speaker’s honoraria and travel assistance from Baxter Healthcare. The remaining authors have no competing financial interests to declare.

**REFERENCES**


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