In the present study, we evaluated the effects of once-weekly mupirocin application to catheter exit sites on Staphylococcus aureus and coagulase-negative staphylococcus (CNS) colonization and investigated the resistance of those bacteria to methicillin (MeR) and mupirocin (MuR).

We enrolled 36 continuous ambulatory peritoneal dialysis (CAPD) patients (mean age: 55.1 ± 1.4 years) into the study. The patients (men/women: 21/15) had been applying mupirocin to the catheter exit site once weekly before the start of the study (mupirocin treatment duration: 3.1 ± 2.0 years). During the study period, swabs were taken monthly from the nares, axillae, inguinal area, and catheter exit site. The swabs were inoculated on blood plates. Methicillin and mupirocin susceptibility were tested by disc diffusion according to the interpretative criteria of the National Committee for Clinical Laboratory Standards.

We evaluated a total of 144 cultures. Among CNS isolates, the MuR was 66%, and the MeR was 38.8%. At the start of the study period, 3 patients were S. aureus nasal carriers. In nasal swabs, no MeR S. aureus was identified, and only 1 MuR S. aureus was found.

Once-weekly application of mupirocin at the exit site in CAPD patients led to comparable rates of colonization by MuR S. aureus as did thrice-weekly or more frequent application. Clinical results showing high mupirocin and methicillin resistance in CNS are controversial.

Key words
Continuous ambulatory peritoneal dialysis, mupirocin, mupirocin resistance

Introduction
Staphylococcus species are the most frequent cause of peritonitis and exit-site infection (ESI) in continuous ambulatory peritoneal dialysis (CAPD) patients (1,2). The rate of colonization with S. aureus in peritoneal dialysis (PD) patients is reported to be around 60%, and rates of infection with that micro-organism have increased threefold in the last decade (3). Peritonitis, the major complication of PD, causes prolonged hospitalization, economic loss, and compulsory hemodialysis procedures (1,3).

To prevent infections associated with S. aureus, several antibiotics—for example, bacitracin, sodium fusidate, rifampicin, and neomycin sulfate—have been employed (4,5). However, those agents have fallen out of use today because of resistance problems and potential side effects. Recently, mupirocin has been extensively used to prevent S. aureus infections in PD patients.

Mupirocin (also known as pseudomonic acid) is produced by Pseudomonas fluorescens. It inhibits protein synthesis by binding to isoleucyl-transfer RNA synthase and is a potent agent against staphylococci (6). The drug cannot be administered systemically because of its rapid degradation into inactive metabolites in vivo. Thus, applying mupirocin intranasally or at the exit site is recommended (4,5).

Currently, the most popular regimen for mupirocin application at the exit site is 3 – 5 times weekly. Mupirocin has been found to eliminate 90% – 100% of colonizations and 50% of ESIs caused by the same...
micro-organisms (5). Mupirocin, which was first used in 1980, and whose first case of resistance was reported in 1987, is recommended for short periods of use. No further studies in CAPD patients of once-weekly mupirocin application at the exit site are available to show whether resistance rates and occurrence of infection are reduced under that regimen.

In the present study, we evaluated *S. aureus* and coagulase-negative staphylococci (CNS) carriage and the resistance of those bacteria against methicillin and mupirocin with weekly application of mupirocin to the catheter exit site in CAPD patients.

**Patients and methods**

We enrolled 36 CAPD patients into the study. The patients had been under CAPD treatment for 3.1 ± 2.0 years (range: 1 – 9 years) and had been applying mupirocin (Bactroban: GlaxoSmithKline, Istanbul, Turkey) to the exit site once weekly for 2.2 ± 0.8 years (range: 1 – 3.1 years). Table I gives demographic data about the patient population.

Patients were seen monthly, and at each visit, swabs were taken from the nares, axillae, inguinal area, and exit site. The swabs were inoculated on blood agar plates, and incubated for 18 – 24 hours at 37°C. The plates were then evaluated for the growth of gram-positive cocci, and the tube coagulase test was applied to catalase-positive colonies to discriminate *S. aureus* and CNS. The colonies were inoculated onto Mueller–Hinton agar plates so that methicillin (oxacillin) and mupirocin sensitivity could be determined by the disk diffusion method. Isolates with an inhibition zone ≥18 mm (CNS) or ≥13 mm (*S. aureus*) around the oxacillin disk (1 µg) were considered sensitive according to the criteria of the National Committee for Clinical Laboratory Standards (7). Isolates of CNS and *S. aureus* with inhibition zone ≥14 mm around the mupirocin disk (5 µg) were also accepted as sensitive.

Statistical analysis was carried out using descriptive statistics.

**Results**

For CNS, resistance to mupirocin (MuR) was found in 66% of all isolates and to methicillin (MeR) in 38.8%. In 3 patients, nasal smears contained *S. aureus*. None of the *S. aureus* isolates exhibited MeR, but 1 showed MuR. Growth of *S. aureus* was not detected in any of the exit-site cultures. Tables II and III summarize the results of the study.

**Discussion**

Peritoneal dialysis is the most effective mode of treatment in end-stage renal failure patients. Infections by *S. aureus* are the major cause of morbidity and hospitalization in dialysis patients. Mupirocin has been extensively used to prevent *S. aureus* infection in patients undergoing PD. Recent studies indicate that mupirocin is an effective antibacterial agent against *S. aureus* colonization and infection (2,7).

Because mupirocin is a topical agent, a formal definition of mupirocin sensitivity has not been established by the National Committee for Clinical Laboratory Standards. However, several studies have set sensitivity limits for mupirocin. Some researchers investigated the results of mupirocin application on the nasal mucosa and at the exit site daily or 3 – 5 times weekly. At the end of the studies, no difference was observed between the two groups (application daily or 3 – 5 times weekly) with regard to effectivity against CAPD-related infections (4,5,7,8). In a previous study,

<table>
<thead>
<tr>
<th>TABLE I</th>
<th>Demographic data for the study patients (n = 36)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [years; mean±SD (range)]</td>
<td>55.1±8.7 (42–81)</td>
</tr>
<tr>
<td>Men/women (n)</td>
<td>21 / 15</td>
</tr>
<tr>
<td>Causes of renal failure (n)</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>11</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>5</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>4</td>
</tr>
<tr>
<td>Polycystic kidney disease</td>
<td>3</td>
</tr>
<tr>
<td>Other</td>
<td>3</td>
</tr>
<tr>
<td>Unknown</td>
<td>10</td>
</tr>
<tr>
<td>Duration of peritoneal dialysis [years; mean±SD (range)]</td>
<td>3.1±2.0 (1–9)</td>
</tr>
<tr>
<td>Duration of mupirocin application [years; mean±SD (range)]</td>
<td>2.2±0.8 (1–3.1)</td>
</tr>
</tbody>
</table>
we found that weekly application of mupirocin to the exit site also reduced peritonitis and ESIs at rates of 48% and 86% respectively—statistics that are roughly equivalent to those from studies employing more frequent use of mupirocin (9). Annigeri et al. (10) reported the emergence of high-level MuR S. aureus in CAPD patients after 4 years of continuous use of mupirocin in a small number of patients in their PD unit. High-level MuR S. aureus were isolated from 3% of the total study population and from 15% of all S. aureus isolates.

In the present study, we found that the colonization rate by S. aureus in CAPD patients was 2%. On the other hand, MuR among those isolates was 33%. Among all the isolates obtained in the present study, the overall rate of MuR was 0.7%. The possible reason for a MuR rate as high as 33% is that we found only 3 S. aureus isolates, and only 1 showing MuR. Longer patient follow-up may yield more realistic results in the future.

In the present study, none of the S. aureus isolates showed MeR. However, the CNS strains showed high rates of MuR and MeR. Among 113 CNS isolates, 95 (66%) showed MuR, and 56 (38.8%) showed MeR. In a neonatal intensive care unit, Zakrzewska–Bode et al. (11) reviewed the effects of 5 years of mupirocin use around central venous catheter entry sites and found that 42% of clinical isolates were MuR CNS. The investigators reported a decrease in MuR to 13% after abandoning the antibiotic. We were not able to find a study in the literature that discussed the effect of the frequency of mupirocin application in CAPD patients on development of MeR and MuR in CNS isolates.

Conclusion
Given our findings, MeR and MuR CNS may further complicate CAPD infections in the near future. Studies about mupirocin should therefore take into account resistance not only in S. aureus, but also in CNS strains. They should also look at the CAPD infections associated with both micro-organisms.

Once-weekly use of mupirocin at the exit site in CAPD patients led to rates of MuR S. aureus emergence that were comparable with thrice-weekly and more frequent application. Clinical results concerning high mupirocin and methicillin resistance in CNS are controversial.

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Aureus exit-site infections and peritonitis in CAPD patients by local application of mupirocin ointment at the catheter exit site. Perit Dial Int 1998; 18:261–70.


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Relapsing peritonitis is usually attributable to persistent catheter infection, assuming that the appropriate antibiotic is used to treat each time and that intra-abdominal pathology has been ruled out. Removal of the peritoneal dialysis (PD) catheter frequently is curative. We describe here a case in which partial removal of a presternal catheter resulted in resolution of relapsing peritonitis.

Key words
Relapsing peritonitis, presternal catheter, titanium connector

Case report
A 62-year-old African–American man with end-stage renal disease and mild liver disease secondary to hepatitis C had been on continuous ambulatory peritoneal dialysis (CAPD, 4 exchanges daily) since August 1999. In October 2002, he developed a first episode of peritonitis with *Acinetobacter baumannii* and typical signs and symptoms of CAPD-associated peritonitis. The peritonitis resolved with 2 weeks of therapy with intraperitoneal (IP) cefazolin and tobramycin. No exit-site infection was evident.

Within a week of antibiotic cessation, peritonitis relapsed with the same species of organism. This second episode of peritonitis was treated with IP tobramycin alone because of cefazolin resistance. Symptoms resolved quickly, but IP tobramycin was continued for 4 weeks.

A third episode of peritonitis with the same species occurred within a week of tobramycin discontinuation. Given the lack of any obvious signs of catheter infection or intra-abdominal pathology, another 2-week course of IP tobramycin, together with oral levofloxacin, was instituted with quick resolution of signs and symptoms. Oral levofloxacin was continued for 2 months. During that 2-month period, the lumen of the catheter was twice locked for 12 hours with 2 mg tissue plasminogen activator (tPA), 2 weeks apart. However, a fourth episode of peritonitis occurred shortly after antibiotic withdrawal. On that occasion, the chest part of the presternal catheter and the titanium adapter were surgically replaced. Oral levofloxacin was given for 1 month. Continuous ambulatory peritoneal dialysis was continued post procedure.

Cultures from the dialysate, the catheter tip, the titanium adapter joint spaces, and the exit site were obtained at the time of surgical removal of the catheter. *Acinetobacter baumannii* grew only from the swab obtained from the titanium joint spaces. The surgical approach resolved the peritonitis, and the patient was still free of peritonitis at 7 months after surgery (the time of writing).

Presternal catheters have two segments: chest and abdominal. The two segments are joined at a titanium adapter at the time of insertion. The titanium joint is located under the skin below the costal margin and is never routinely replaced. The present case provides an example of an unusual location where bacteria can reside and become the source of continued contamination of the peritoneal cavity.

Discussion
Peritonitis has long been known as a complication of peritoneal dialysis access. The current rate of peritonitis at our center is 1 episode per 48 patient–months, based on 25 years of experience (Figure 1).
Determining the source of infection—whether it be nasal carriage, exit-site infection, tunnel infection, biofilms, intra-abdominal pathology, or hematogenous spread—has been the subject of much literature. The present case report illustrates an unusual site of infection and a new approach to preservation of an access.

The most common pathogens of peritonitis have been gram-positive *Staphylococcus aureus* and *S. epidermis*. However, gram-negative infections—notably, *Pseudomonas aeruginosa*—have been increasing in incidence. Our case involved *Acinetobacter baumannii* infection. That bacterium has an annual incidence in peritoneal cultures of about 0% – 4% (Table I).

Peritonitis usually presents as abdominal pain or fever with cloudy peritoneal fluid. To make a diagnosis of peritonitis, the physician should be ever-vigilant and should send the dialysate effluent for differential cell count, Gram stain, and culture. Shorter dwell times (that is, <4 hours) have been theorized to lead to negative cultures and reduced cell counts. In peritonitis, the white blood cell count in the peritoneal fluid is usually >100 cells/mm$^3$, but can be less in a small percentage of cases. Low cell counts should not preclude a work-up, and the above-mentioned lab work should be undertaken if the patient has presenting signs and symptoms. Empiric therapy should then be started. Subsequent therapy is guided by organism identification and sensitivities at 24 – 48 hours.

The emergence of vancomycin-resistant organisms has influenced empiric antibiotic regimens. In the present case, our initial empiric regimen was a first-generation cephalosporin and an aminoglycoside (because preservation of residual renal function was not a concern in this particular patient). We then adjusted our antibiotic

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>%</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram-positive bacteria</td>
<td>55–80</td>
<td></td>
</tr>
<tr>
<td>Coagulase-negative <em>Staphylococcus</em></td>
<td>35–70</td>
<td></td>
</tr>
<tr>
<td>Coagulase-positive <em>Staphylococcus</em></td>
<td>10–25</td>
<td></td>
</tr>
<tr>
<td>Other gram-positive bacteria</td>
<td>N/A</td>
<td>3–15</td>
</tr>
<tr>
<td><em>Streptococcus</em> species</td>
<td></td>
<td>3–10</td>
</tr>
<tr>
<td><em>Enterococcus</em></td>
<td></td>
<td>0–4</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
<td>17–30</td>
</tr>
<tr>
<td><em>Escherichia coli</em></td>
<td></td>
<td>4–10</td>
</tr>
<tr>
<td><em>Pseudomonas</em> species</td>
<td></td>
<td>5–10</td>
</tr>
<tr>
<td><em>Klebsiella</em> species</td>
<td></td>
<td>1–5</td>
</tr>
<tr>
<td><em>Acinetobacter</em> species</td>
<td></td>
<td>0–4</td>
</tr>
<tr>
<td><em>Enterobacter</em> species</td>
<td></td>
<td>0–3</td>
</tr>
<tr>
<td><em>Serratia</em> species</td>
<td></td>
<td>0–3</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
<td>0–6</td>
</tr>
</tbody>
</table>

Adapted, with permission from Elsevier Health Sciences, from: Rector and Brenner’s The Kidney. 6th ed. Philadelphia: WB Saunders; 2000: 2486. [Table 58–11]
regimen per sensitivities and stopped the cefazolin. No obvious exit-site or tunnel infection was present; but, after several reoccurrences of peritonitis with the same organism, attempts shifted to salvage of the catheter.

As with many dialysis patients, access was a major concern. Switching to hemodialysis was not an option for this patient and thus attempts were made to preserve the current catheter. Intraluminal tPA administration was attempted on two separate occasions, but those attempts were subsequently followed by another episode of peritonitis.

A decision to replace the presternal portion of the catheter and to attempt also to change the titanium connector was then taken. Significant literature is available regarding simultaneous replacement of the presternal portion of the catheter in patients with poor access, but the literature does not mention any defined protocol for replacing the titanium connector.

After removal of the presternal portion of the catheter and the titanium connector, the relapsing peritonitis resolved. The patient is symptom-free to date. Cultures from the titanium connector returned positive for \textit{A. baumannii}. That finding confirmed a previously undocumented site for infection.

**Conclusion**

To maximize the chance of salvaging a peritoneal catheter in individuals with relapsing peritonitis, consider the possibility of infection residing in the titanium adapter. In our case, replacement of the titanium connector resolved a difficult clinical situation.

**References**


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The Recommendations from the International Society for Peritoneal Dialysis for Peritonitis Treatment: A Single-Center Historical Comparison

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The antibiotic treatment currently recommended by the International Society for Peritoneal Dialysis (ISPD) for peritonitis consists of a combination of a first- and a third-generation cephalosporin. The schedule formerly recommended combined a first-generation cephalosporin and an aminoglycoside. No comparison between the treatment schedules has been performed until now. We compared the effectiveness of these two regimens in peritoneal dialysis–related peritonitis at our center.

From January 1999 to April 2000, we followed 107 patients in our PD clinic (period 1: 47% men; 32% with diabetes; mean age: 52 ± 13 years). We followed a similar number of patients from January 2002 to July 2003 (period 2: 109 patients; 54% men; 51% with diabetes; mean age: 56 ± 18 years). In each period, diagnosis and treatment of peritonitis were based on the recommendations of the ISPD as earlier described.

Negative culture rates were similar in period 1 and period 2 (32% vs. 30%). In both study groups, the bacteria that most commonly caused peritonitis were Staphylococcus epidermidis (period 1: 41%; period 2: 39%) and S. aureus (period 1: 27%; period 2: 18%). Gram-positive infections occurred in 59% of patients during period 1 and in 57% during period 2. Gram-negative infections occurred in 16% of patients during period 1 and in 18% during period 2. We observed no significant difference in the peritonitis cure rate from period 1 to period 2 (78% vs. 83%; chi-square: 0.98; p = 0.3), but changes in the primary antibiotic schedule were necessary in 4 patients in period 1 as compared with 1 patient in period 2. The rates of catheter removal were not significantly different during the two periods (period 1: 14%; period 2: 5%; chi-square: 2.5; p = 0.11). Mortality was also not significantly different during the two periods (period 1: 7%; period 2: 5%; chi-square: 0.23; p = 0.62).

The two antibiotic schedules were equally effective in the treatment of peritonitis. Cost-effectiveness, impact on residual renal function, and potential development of bacterial resistance must be considered when selecting the antibiotic schedule for peritonitis treatment.

Key words
Peritonitis, antibiotic treatment

Introduction
Peritonitis is one of the major complications of peritoneal dialysis (PD) and remains the primary reason that patients switch from PD to hemodialysis (1). The reported incidence of peritonitis varies from center to center, in the range of 1 episode every 24 – 60 patient-months (2).

Controversy regarding the use of vancomycin (the most commonly used antibiotic during the early years of PD), based on the observation (3) of PD-associated peritonitis with vancomycin resistant enterococci (VRE), prompted the proposal by the International Society for Peritoneal Dialysis (ISPD) in the late 1990s of a vancomycin-free treatment schedule (4). That treatment schedule combined a first-generation cephalosporin and an aminoglycoside (5). Because of the cumulative understanding of the importance of re-
sidual renal function (RRF), the ISPD Advisory Com-
mittee on Peritonitis Management revised the proto-
col in the year 2000 (6), substituting a third-generation
cephalosporin for the aminoglycoside.

Despite the recommendations by the Advisory
Committee (5,6), peritonitis treatment schedules still
vary from center to center, and the clinical results of
the schedules are not well established (7). Moreover,
a comparison between the two latest sets of ISPD
recommendations for peritonitis treatment has not been
performed until now.

Patients and methods
Our retrospective study was performed in a single
university-based PD center. Only patients on continu-
ous ambulatory PD who were using standard glucose
solutions and who were performing 4 exchanges daily
were included.

Two distinct treatment schedules for peritonitis
were used in our patients during two different time
periods. From January 1999 to April 2000 (period 1),
peritonitis treatment was performed according to the
1996 recommendations of the ISPD (5). During that
study period, we followed 107 patients (47% men;
32% with diabetes; mean age: 52 ± 13 years) for a
cumulative 902 patient–months of experience
(Table I). From January 2002 to July 2003 (period 2),
we changed our peritonitis treatment schedule to fol-
low the recommendations in the ISPD 2000 update.
During that period (period 2), we followed 109 pa-
tients (54% men; 51% with diabetes; mean age: 56 ±
18 years), for a total of 1200 patient–months of expe-
rience (Table I).

In both study periods, peritonitis was defined as
the presence of two clinical signs of peritonitis (ab-
dominal pain, fever, or diarrhea, or a combination)
and an increased peritoneal dialysate white blood cell
count (>100 cells/mm³, with 50% neutrophils). Upon
diagnosis, treatment was initiated. Preceding intraperi-
toneal treatment, peritoneal effluent was aseptically
collected for leukocyte count, Gram stain, and cul-
ture. Patients were trained to inject antibiotics into
their PD fluid bags and were allowed to return home
or were hospitalized if clinically indicated.

In period 1, the treatment schedule was gentami-
cin (loading dose of 80 mg, followed by a mainte-
nance dose of 40 mg daily, always in the night dwell)
combined with cephalothin (loading dose of 1 g, fol-
lowed by a maintenance dose of 250 mg in every ex-
change). In period 2, the schedule was cephalothin
/loading dose of 1 g, followed by a maintenance dose
of 1 g in the night dwell) and ceftazidime (loading
dose of 1 g, followed by a maintenance dose of 1 g in
the night dwell).

Treatment was standardized at 14 consecutive
days, except in the case of methicillin resistant Staph-
ylococcus aureus and Pseudomonas species, in which
case, treatment was extended for another 7 days. An-
tibiotic adjustments were made according to the ini-
tial clinical response and culture results.

For the present analysis, therapy was considered
successful if all signs and symptoms of peritonitis were
eliminated by the prescribed treatment, and if the pa-
tient remained infection-free for 2 weeks following
the end of treatment. The effectiveness of the drugs in
the study was defined by the clinical and bacterio-
logic response of the patient to each infecting organ-
ism. Statistical analysis was carried out using the
chi-square test. The null hypothesis was rejected at
p values of less than 0.05.

Results
In period 1, 55 episodes of peritonitis occurred in
107 patients (1 episode for every 16.4 patient–
months); in period 2, 46 episodes occurred in 109 pa-
tients (1 episode for every 25.5 patient–months; Table I). No significant differences in the distribution
of bacteria species were encountered between the two
periods. Similarly, the clinical characteristics of the
study populations were not different (Table I).

In both study groups, the bacteria most commonly
causing peritonitis were S. epidermidis (period 1: 41%; period 2: 32%) and S. aureus (period 1: 27%;
period 2: 18%). Gram-positive infections occurred in
59% of patients during period 1, and in 57% during

| TABLE I Clinical characteristics of patients followed in the dialysis unit from January 1999 to April 2000 (period 1) and from January 2002 to July 2003 (period 2) |
|-----------------|-----------------|-----------------|
| **Period 1**    | **Period 2**    |
| Patients (n)    | 107             | 109             |
| Age (years)     | 52±13           | 56±18           |
| Men (%)         | 47              | 54              |
| With diabetes (%) | 32           | 54              |
| Time on dialysis (months) | 21±12           | 23±14           |
| Cumulative experience (patient–months) | 902             | 1200            |
| Peritonitis episodes/patient–month | 1/16           | 1/25            |
period 2. Gram-negative infections occurred in 16% of patients during period 1, and in 18 during period 2. Documented oxacillin resistance occurred in 23% of micro-organisms isolated in period 1 as compared with 4% in period 2. The prevalence of negative cultures was similar in periods 1 and 2 (32% vs. 28%).

Catheter removal was not significantly different in the two periods (period 1: 14%; period 2: 5%; chi-square: 2.5; \( p = 0.11 \); Figure 1). Causes of catheter removal in period 1 included \textit{S. aureus} infection in 3 patients, gram-negative organisms in 4 patients, and a fungus in 1 patient. In period 2, 2 catheters were removed for fungal peritonitis.

Most importantly, we observed no significant difference in the cure rate for the two periods (period 1: 78%; period 2: 83%; chi-square: 0.98; \( p = 0.3 \); Figure 1). However, more changes in the primary antibiotic schedule (not statistically significant) were necessary in patients during period 1 (\( n = 4 \)) as compared with period 2 (\( n = 1 \)). Mortality was also not significantly different between the two periods (period 1: 7%; period 2: 5%; chi-square: 0.23; \( p = 0.62 \); Figure 1). During period 1, 4 patients died: 1 from fungal peritonitis, 1 from \textit{S. epidermidis} peritonitis, 1 from polymicrobial peritonitis, and 1 following peritonitis with a negative culture. In period 2, 2 patients died: 1 from fungal infection, and 1 from \textit{S. aureus} infection.

Discussion

Many different antimicrobial agents have been used to treat PD peritonitis. The ISPD Advisory Committee on Peritonitis Management met in 1987 (and again in 1989, 1993, 1996, and 2000) with the aim of reviewing the experiences reported in the literature and of formulating recommendations based on the review. Because the Committee did not suggest that its recommendations represented the only acceptable way to manage PD patients with peritonitis, a variety of regimens have been proposed over the years. In the present study, we describe for the first time a comparison between the two most recent (and perhaps most utilized) treatment schedules recommended by the ISPD. The chief finding of our study was that, in a large single-center population, both schedules were equally effective in treating PD-associated peritonitis.

The recent emergence of vancomycin resistance (3) has created a therapeutic dilemma of international proportions. As a result, major modifications to the guidelines were proposed in 1996 (5), with the main alteration being the restriction of the use of vancomycin to cases with documented oxacillin resistance. In the experience described in the present study, restricting the use of vancomycin to cases in which it was specifically indicated produced satisfactory clinical results.

In the series based on our experience during 1999 and 2000, our treatment success rate met the recommendations of the ISPD and was comparable to previous reports that showed better results with cephalosporin than with vancomycin (8). Concerns regarding that treatment option as compared with treatment in period 2 were the relatively high need for catheter removal and changes in the primary antibiotic schedule. However, those differences represent only a trend and did not reach statistical significance.

In 2000, new changes were introduced in the ISPD treatment recommendations. The key alteration was to avoid the use of aminoglycosides in patients with RRF. The change was stimulated by the observation that use of aminoglycosides was associated with faster decline of RRF in PD patients (9) and that a large body of evidence showed that RRF was strongly related to better outcomes in PD patients (10,11). According to the view of the ISPD Committee, aminoglycosides should not be used as first-line therapy in patients with significant RRF; instead, a third-generation cephalosporin should be used in combination with the first-generation cephalosporin.

Our experience with the revised schedule was also satisfactory, and the clinical results achieved were, in
general, similar to those stemming from the use of the 1996 recommendations. Indeed, the only other study evaluating the efficacy of the new protocol also showed very good clinical results, although the analysis was performed only in a pediatric population (12). The negative aspects of the treatment schedule are higher costs and the potential development of resistance from the combination of a first- and a third-generation cephalosporin.

The recent findings of Baker et al. (13)—which showed, in a small cohort of patients, no evidence of an accelerated decline in RRF when using an empirical regimen containing aminoglycosides for peritonitis—underlines the need for future studies analyzing the true value of avoiding aminoglycosides to protect RRF in PD patients. One important limitation of our study that needs to be clarified is the lack of analysis of the impact of RRF in both treatment schedules.

Conclusion

Our historical retrospective analysis compared the two most recent recommendations for peritonitis treatment in PD and showed that the clinical results were equally good under the 1996 and the 2000 guidelines suggested by the ISPD’s Advisory Committee on Peritonitis Management. Thus, the characteristics of the local flora, regional cost-effectiveness, possible impact on RRF, and potential development of bacterial resistance must be considered when selecting an antibiotic schedule for peritonitis treatment.

References


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