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Diagnosis and Management of Enteric Disease and Abdominal Catastrophe in Peritoneal Dialysis Patients With Peritonitis

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Diagnosis and Management of Enteric Disease and Abdominal Catastrophe in Peritoneal Dialysis Patients With Peritonitis

Mark D. Faber and Jerry Yee

Peritoneal dialysis (PD)–associated peritonitis rates have decreased significantly in recent years, especially *Staphylococcus epidermidis* and *Staphylococcus aureus* infections. Rates of gram-negative, polymicrobial, and fungal peritonitis have remained steady. The reported mortality of gram-negative and polymicrobial peritonitis varies widely (4%-50%). Most likely, the reason for this variability is that prognosis depends on the underlying etiology more than the specific microorganisms isolated. Gram-negative, polymicrobial, and fungal infection have variable association with documented visceral disease, and the highest mortality occurs in reports with the highest prevalence of intra-abdominal pathology. The odds ratio of death in PD patients with documented abdominal catastrophe and peritonitis is reported to be 20:1 compared with all other causes. Further reductions in PD-associated peritonitis mortality are likely to depend on earlier diagnosis and better management of intra-abdominal pathology. Presentation with hypotension, sepsis, lactic acidosis, and/or elevation of peritoneal fluid amylase should raise immediate concern for "surgical" peritonitis. Suspicion for visceral disease should also be high in patients with gram-negative, polymicrobial, and fungal infection or those who fail to improve rapidly as judged by clinical signs and symptoms, cell counts, and repeat cultures. Nonlocalizing physical examination and negative or nonspecific results of abdominal computed tomography do not rule out serious intra-abdominal disease. Immediate initiation of broad antibiotic coverage including for anaerobic infection is indicated when bowel pathology is suspected. Urgent surgical consultation, with active discussion and participation by the nephrologist, is advisable when visceral pathology is suspected and the patient is unstable or fails to improve rapidly.

Index Words: Peritoneal dialysis; Peritonitis; Enteric disease; Gram-negative peritonitis; Polymicrobial peritonitis

"It was the best of times; it was the worst of times." Were Dickens a nephrologist, this would likely be his current impression of peritoneal dialysis (PD)-associated peritonitis. Twenty-five years ago, most continuous ambulatory peritoneal dialysis programs reported peritonitis rates in excess of 1 to 2 episodes per patient year.[1,2] By contrast, recent International Society for Peritoneal Dialysis guidelines[3] state that all programs should be able to achieve peritonitis rates of less than 0.67 infections per patient year at risk. Many programs report rates of less than 0.3 infections per year.[4,5] Decreases in the incidence of *Staphylococcus epidermidis* infection can be directly traced to successive advances in connectology (eg, "Y" sets, twin bag sets, spikeless connections, sterile connection devices, and cyclers).[6,7] Simultaneously, the widespread use of anti-*Staphylococcus* prophylaxis (eg, nasal or exit-site mupirocin or exit-site gentamicin cream)[8–11] has dramatically reduced the risk of *Staphylococcus aureus* peritonitis and catheter infection. Unfortunately, mortality because of peritonitis has not decreased in parallel and still accounts for more than 15% of reported deaths.[12,13] The mortality of *S epidermis* peritoneal infection has been reported as less than 1 percent,[12] but deaths after *S aureus* peritonitis have been reported in 3.4%[14] to 15%[13] of episodes. Moreover, although *Staphylococcus* (and in some reports *Pseudomonas*) infections have indeed decreased,[15] the incidence of the peritoneal infections associated with the highest reported mortality (gram-negative,[14] enterococcal,[14] fungal, and polymicrobial[13,16] peritonitis) has not decreased. Newman et al[17] compared organism-specific peritonitis rates during 1988 to 1996 to

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the period from 1996 to 2000. The most impressive decrease was in *S aureus* peritonitis (0.136-0.053 episodes per patient year). Other non-*Streptococcus* gram-positive infections also decreased (0.281-0.232 episodes per patient year). In contrast, the rates of enteric, polymicrobial, *Streptococcus*, *Candida*, and *Enterococcus* infections each remained stable or increased. The explanation for the poorer outcomes associated with these particular infections is incomplete. It is likely that bacterial virulence factors are responsible in some infections (especially *S aureus*, fungal, and *P aeruginosa*), whereas the association with enteric infection and "abdominal catastrophe"[16,18] probably underlies the virulence of most of the other organisms listed.

The most difficult decision that a physician caring for a PD patient with peritonitis must make is whether or not to request (or insist) that a surgical colleague explore the patient for the presence of intra-abdominal pathology. Despite its invasive nature and inherent risk, laparoscopy or laparotomy in judiciously selected patients may be the key opportunity to further reduce peritonitis-associated deaths in this population. This article will use the terms abdominal catastrophe; surgical peritonitis; intra-abdominal, enteric, or visceral pathology; disease; injury; or perforation interchangeably and in reference to serious intra-abdominal disease typically considered to be an indication for surgical intervention. In contrast, "enteric organisms" as defined by Harwell et al[18] are microorganisms typically residing in the gastrointestinal tract and include *Klebsiella*, *Enterobacteriaceae*, *Serratia*, *Escherichia coli*, *Proteus spp*, *Morganella spp*, *Citrobacter spp*, *Enterococcus spp*, and *Bacteroides spp*. They also include organisms not always recognized as enteric in origin including viridans streptococci and *Torulopsis*. Published experience makes it abundantly clear that peritonitis associated with visceral injury in PD patients entails high mortality,[16] although the issue is confused by various reports defining "enteric," "intrinsic," or "endogenous" peritonitis according to the results of dialysate cultures rather than documented visceral injury or infection.

Other new challenges face PD patients and the physicians caring for them today. The increasing incidence of antibiotic-resistant microorganisms (including methicillin-resistant *S aureus* and *S epidermis* and vancomycin-resistant *Enterococcus*) further complicates empiric therapy of peritonitis.[15,19] This may increase the risk of poor outcomes if effective therapy is delayed by incorrect initial antibiotic choice. This review will explore in detail the overlapping clinical issues that arise in PD patients with gram-negative peritonitis, polymicrobial peritonitis, and suspected or presumed "abdominal catastrophe" and then outline a treatment approach. The reader is referred to several recent excellent reviews and treatment guidelines that address routine aspects of prevention, diagnosis, and treatment of peritonitis.[3,20–25]

Gram-Negative Peritonitis

Overall reported mortality directly associated with a PD-related peritonitis episode has generally been reported to range from 2% to 6% [13,26] (Table 1). Multiple reports make it clear, however, that gram-negative peritonitis contributes disproportionately to mortality. Bunke et al[27] reported 6.5% mortality in PD patients with *Pseudomonas* peritonitis (*v* 2.4% in all other cases). They later compared the mortality associated with non-*Pseudomonas* gram-negative peritonitis to that of gram-positive infection.[14] Although mortality of non-*Pseudomonas* gram-negative infection was higher than for *S epidermidis* peritonitis (3.7% *v* 0.8%), it was similar to that of *S aureus* (3.4%) and lower than that of *Enterococcus spp* (7.4%). Rates of catheter removal and transfer to hemodialysis were somewhat higher for non-*Pseudomonas* gram-negative infection than for *S aureus*, especially in the absence of exit-site or tunnel infection. Fried et al[12] reported that death resulted in 9.5% of all gram-negative or fungal episodes. Similarly, Perez-Fontan et al[13] reported 19.3% mortality in association with infection by "enteric" organisms. Although not reporting organism-specific mortality rates, Kern et al[16] reported an odds ratio of death of 20.7:1 (95% confidence interval, 2.40-178.5) in patients with gram-negative peritonitis compared with gram-positive infection.

Table 1. Reported Outcomes of Gram-negative PD-Associated Peritonitis

Reference No.	*Author, Year*	*Microbiology*	*Gram-Negative Mortality*	*Confirmed Abdominal Pathology*	*Reference Group Mortality*	*Other Gram-negative Long Term Outcomes*
[27]	Bunke, 1995	*Pseudomonas spp*	6.5%	Not reported	Non-*Pseudomonas* gram-negative (2.4%)	Catheter loss 61%, switch to HD 26%
[12]	Fried, 1996	All gram-negative or fungal	9.5%	Not reported	*S epidermidis* (0.5%) Other (2.5%)	
[14]	Bunke, 1997	Non-*Pseudomonas* Gram-negative	3.7%	3/136 (2.2 %)	*S epidermidis* (0.8%) *S aureus* (3.4%) *Enterococcus spp* (7.4%)	Catheter loss 30%, switch to HD 14%
[16]	Kern, 2002	Single gram-negative	Odds Ratio 20.7 (CI 2.4–178.5)	56%	Gram-positive	Catheter loss 30%, switch to HD 14%
[51]	Prasad, 2003	Single gram-negative (66% "fecal" origin)	20%	Not reported	Single gram-positive (10%) (53% "fecal" origin)	Catheter loss 37% Switch to HD 8.9%
[13]	Perez-Fontan, 2005	Enterobacteriaciae *Pseudomonas spp* Other gram-negative	4.7% 11.1% 0%	Not reported	*S epidermidis* (0.5%) *S aureus* (15.2%) *Enterococcus spp* (7.4%)	

Abbreviation: HD, hemodialysis.

Polymicrobial Peritonitis

Szeto et al[28] reported on 140 episodes of polymicrobial peritonitis in 112 patients (Table 2). The full spectrum of possible microbiological mixes was represented, including only gram-positive organisms (28%), mixed gram-positive and gram-negative infection (24%), only non-*Pseudomonas* gram-negative organisms (9%), *Pseudomonas* (11%), and fungal infections (21%). Ninety episodes (64%) responded to antibiotic therapy alone. Approximately 70% of patients remained on or returned to peritoneal dialysis after resolution. The clinical algorithm used in Szeto et al's report was to consider laparotomy and/or catheter removal only if infection failed to respond after 10 days of appropriate antibiotic therapy. Thus, although the authors assumed that all of these cases were caused by some type of abdominal visceral perforation, ex-

Table 2. Reported Outcomes of Polymicrobial PD-Associated Peritonitis

Reference No.	*Author, Year*	*Microbiology*	*Polymicrobial Mortality*	*Confirmed Abdominal Pathology*	*Reference Group Mortality*	*Long-Term Outcomes*
[32]	Van der Reijden, 1988	"Enteric" (2 or more gram-negatives, most with Bacteroides)	57% by 13 days 72% at 2 months			
[29]	Holley, 1992	Enteric and nonenteric	2.6% of patients	6.9% of episodes		Catheter removal 40% Hemodialysis transfer 16%
[30]	Kiernan, 1995	Gram-positive, Gram-negative, And/or fungal	5% immediate 13.7% at 6 mos	7.5% of episodes		
[33]	Suh, 1996	"Enteric" (2 or more gram-negatives)	13%			33% still on PD 38% develop fungal peritonitis despite prophylaxis
[31]	Kim, 2000	Gram-positive, Gram-negative, And/or fungal	11% at 3 years	7% of patients	Single organism (33% 3 years) No peritonitis (36% 3 years)	70% transfer to HD at last follow-up (33 ± 26 mos)
[28]	Szeto, 2002	Gram-positive, Gram-negative, And/or fungal	9% immediate 15% at 3 months	4.4%		90% response to antibiotics; 70% remain on or return to PD
[13]	Perez-Fontan, 2005	Nonenteric polymicrobial Enteric polymicrobial	4.5% 19.4%		*S epidermidis* (0.5%) *S aureus* (15.2%) *Enterococcus spp* (7.4%)	

Abbreviations: HD, hemodialysis; PD, peritoneal dialysis.

Table 3. Reported Outcomes of PD-Associated Peritonitis With Confirmed Visceral Injury

Reference No.	*Author, Year*	*Microbiology*	*Mortality*
[18]	Harwell, 1997	Single enteric 22/26 Polymicrobial 3/26	46.3% enteric peritonitis 7.5% other peritonitis
[16]	Kern, 2002	Single Gram-negative 58.5% in confirmed visceral injury (11.8% other etiologies of peritonitis) Polymicrobial < 20% OR. of enteric infection 66:1 (CI 7.9–551.3) in patients with gram-negative peritonitis OR of enteric infection 22:1 (CI 1.6–315.1) in patients with fungal peritonitis	OR of death 20.1 (CI 5.4–75.2) with peritonitis because of documented visceral injury, 41.5 (CI 5.5–317.5) with peritonitis because of sepsis

Abbreviations: OR, odds ratio; CI, confidence interval.

ploratory laparotomy was performed in only 8 cases. Intra-abdominal pathology was confirmed in only 3 of these surgical explorations (strangulated hernia, ischemic colitis with perforation, and sigmoid colon carcinoma). Unfortunately, another 13 patients (9%) died before surgical exploration was performed. Of these 13 early deaths, severe peritonitis was the direct etiology in 9. Seven of these 9 underwent postmortem examination, during which only 1 case of bowel pathology (perforated colonic diverticulum) was found. Another 8 patients died subsequently of various causes within 3 months. Thus, although total 3-month mortality was 15%, it is unclear what percentage of these patients had visceral disease and whether earlier operative intervention (laparotomy and/or catheter removal) would have reduced total mortality or that caused directly by peritonitis.

Three earlier, smaller published series of PD patients with polymicrobial peritonitis[29–31] reported similar findings. Specific intra-abdominal pathology was documented in only 7% of cases, although most patients were not specifically investigated. Deaths immediately attributable to peritonitis were infrequent (3.7%), although eventual mortality was still appreciable. For example, Kiernan et al[30] reported only 4 deaths directly attributable to 80 polymicrobial peritonitis episodes but another 7 deaths in the following 6 months (total mortality, 14%). Interestingly, Kim and Korbet[31] reported that patients with polymicrobial peritonitis had higher survival (91% at 3 years) than either patients with single organism peritonitis (67%) or patients without peritonitis (37%). Nevertheless, long-term technique survival was extraordinarily low after polymicrobial infection (30% at last follow up, averaging 33 ± 26 months).

The experience of Kern et al[16] was substantially different, in that abdominal catastrophe was documented in 73% of polymicrobial peritonitis episodes in their report. Mortality for this specific subset was not calculated but was 46.3% for the entire group of patients with abdominal catastrophe. In summary, with the exception of Kern et al's report, the published predictive value of polymicrobial infection for injury of the abdominal viscera is generally low, especially for serious disease requiring operative intervention. Nonetheless, these patients appear to be at high risk over time for either technique failure (primarily attributed to recurrent peritonitis) or mortality.

Peritonitis Because of Visceral Pathology or Abdominal Catastrophe

Van der Reijden et al[32] defined "fecal" peritonitis by the presence of 2 or more gram-negative organisms in dialysate cultures, although most cultures also contained *Bacteroides* species (Table 3). Three patients recovered uneventfully after antibiotic treatment and PD catheter removal. In contrast, 4 others (57%) suddenly deteriorated 1 to 13 days after presentation and died despite even-

tual surgical exploration and treatment for sepsis and bowel perforation. One additional patient survived surgical exploration immediately on identification of anaerobic organisms but died 2 months later. Most of the documented cases were caused by perforation of the sigmoid colon. By using a similar definition, Suh et al[33] reported on 15 patients with "endogenous" peritonitis, representing 7% of all peritonitis episodes. Most of these cases were preceded by severe constipation, and most of the documented cases were caused by ruptured colonic diverticulae. All patients received appropriate antibiotic therapy and antifungal prophylaxis. Three patients required colectomy and colostomy, and another patient required cholecystectomy. There were only 2 deaths (13%), including 1 of the colectomy patients and another who refused surgery. However, only 5 patients ultimately remained on PD. Despite fluconazole prophylaxis, six patients (38%) required catheter removal due to subsequent fungal peritonitis after a mean of 11 days (range, 3-24 days).

A comprehensive report by Harwell et al[18] shows that confirmed visceral injury is not a rare event in the PD population. They found that abdominal catastrophe occurred once in 153.1 patient months in PD patients (cumulative incidence, 11.3% of patients) compared with an estimated once in 10,000 patient months on hemodialysis and 2,892 patient months in kidney transplant recipients. This report also documents the difficulty of assigning the etiology of peritonitis solely by the result of dialysate cultures. The probable cause of each of 354 peritonitis episodes that occurred in 132 patients was determined. Ninety-eight patients (43%) had no peritonitis. A single enteric organism was isolated in 22 of 26 final peritonitis episodes attributable to confirmed visceral injury. Polymicrobial infection was present in only 3 cases. There were 11 instances of ischemic bowel, 3 of gangrenous cholecystitis, 6 of ruptured diverticular disease, 4 of appendicitis, and 1 case of perforated pyloric ulcer. These patients represented 19.7% of all patients with peritonitis and 11% of all patients on PD. Moreover, documented enteric injury accounted for only 32.5% of peritonitis cases caused by enteric organisms, whereas catheter-related infection and technique failure together accounted for 42.5% of infections with "enteric" pathogens.

There is another sobering observation from these authors. Fifty percent of the 26 patients with documented visceral injury died, constituting 33 percent of the episodes with documented enteric disease. This mortality rate is similar to that observed in earlier series of peritonitis resulting from visceral pathology.[34–36] Clearly, although some of the reports cited above may provide support for a sanguine or "wait and see" attitude about apparently stable patients with gram-negative or polymicrobial peritonitis, this is inadequate for patients with discernable intra-abdominal pathology. Harwell's initial report experience was updated in 2002.[16] The update adds considerable new information. Anatomically documented abdominal catastrophe can be segregated into higher- and lower-risk groups, at least in the setting of heightened awareness and surveillance for the condition, coupled with early intervention when suspected. Of the 16 patients who developed documented abdominal catastrophe from 1996 to 2000, no patient with gallbladder or diverticular disease died, and most returned to PD after recovery. One patient with a perforated duodenal ulcer and both patients with strangulated hernias recovered and remained on PD as well. In contrast, despite timely operative intervention, all 5 patients with diffuse ischemic gastritis, enteritis, or colitis died. Two other patients with perforated peptic ulcer also died. Conceivably, the outcome of even the lower-risk group might have been worse without the proactive approach that was adopted.

Diagnosis of Abdominal Catastrophe

A suggested diagnostic approach to PD-associated peritonitis is shown in Figure 1. The initial step is clinical evaluation (history and physical examination) and collection of peritoneal effluent for gram-stain, culture and sensitivity, and amylase concentration. The presence of visceral injury is seldom certain at the time a PD patient presents with peritonitis, with the exception of acute bowel perforation during peritoneoscopic catheter placement.[37] Suspicion may be high earlier for specific clinical disorders, such as mesenteric ischemia in patients with known or suspected

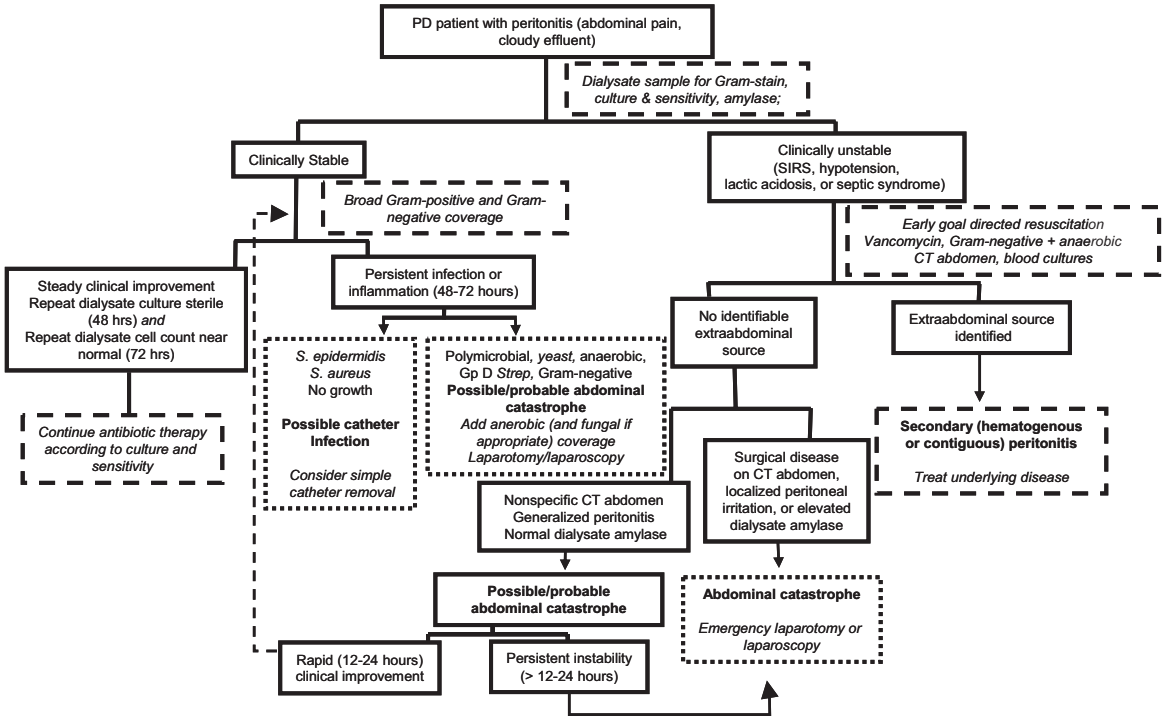

Fig 1. Algorithm for identification and management of suspected visceral disease in PD patients with peritonitis. Perforated box, diagnostic testing or medical therapy decision; dotted box, surgical therapy decision; solid arrow, diagnostic conclusion; perforated arrow, potentially unstable, monitor closely for clinical deterioration.

vascular disease, and typical laboratory features such as elevation of serum lactate and lactate dehydrogenase (LDH). Other clinical disorders, such as appendicitis, cholecystitis, or diverticulitis, may be suspected because of localized peritoneal irritation in the corresponding location. However, this is not a reliable finding (vide infra), and lack of localized abdominal tenderness does not rule out the presence of localized intra-abdominal disease. Presentation with septic shock or bacteremia is also distinctly uncommon and should raise the suspicion of serious intra-abdominal pathology if this occurs. More commonly, the suspicion grows over days as culture results confirming gram-negative, polymicrobial, anaerobic, and/or fungal infection become available. Culture results may suggest but seldom prove the presence of visceral injury. Kern et al[16] reported the odds ratio of a patient having visceral peritonitis as 66:1 in patients with gram-negative or fungal organisms in dialysate culture compared with patients with gram-positive infection. Yet, as discussed earlier, only 56% of gram-negative and 33% of fungal infections were of documented enteric origin. Simultaneously, the patient may fail to improve clinically, peritoneal leukocyte counts may remain elevated, and dialysate gram stain or cultures may continue to show microorganisms. The consequence of late recognition can be disastrous. At worst, the diagnosis is made during a postmortem examination.

There are multiple reasons for the typical delay in diagnosis of abdominal catastrophe in PD patients. First, there is generally a low index of suspicion. Nephrologists and surgeons alike often assume that peritonitis in a PD patient is because of touch contamination or periluminal (tunnel) infection. This is compounded by apparent alterations in PD patients of the natural history of conditions that result in an acute abdomen. The ongoing treatment with antibiotics masks without necessarily eradicating ongoing peritoneal contamination, whereas the presence of peritoneal fluid and the performance of peritoneal

flushes obscure the localizing signs that might suggest bowel pathology. Moreover, common imaging studies have decreased sensitivity and specificity in PD patients. The clinical significance of pneumoperitoneum, a key diagnostic feature of intestinal perforation in the general population, appears to be low in PD patients, unless a large amount of air is present.[38–40] A computed tomography scan is commonly negative in the face of proven abdominal pathology, which may result in further delays because of a false sense of security. Peritoneal fluid amylase[41,42] has been reported to distinguish between routine "non-surgical" peritonitis (mean, 11 IU/L; range, 0-90 IU/L) and that associated with pancreatitis (mean, 540; range, 100-1,140) or bowel perforation (mean, 816; range, 142-1,746). Routine use of this assay in PD patients with peritonitis might speed the diagnosis of serious abdominal pathology but has not been tested prospectively.

Management of Suspected Abdominal Visceral Pathology or Abdominal Catastrophe in PD Patients

Fortunately, the routine gram-negative and gram-positive antibiotic coverage for peritonitis outlined in recent International Society for Peritoneal Dialysis guidelines[3] covers the vast majority of aerobic bacteria likely to be isolated from dialysate even in patients with abdominal catastrophe (Fig 1). The major therapeutic void left by the typical regimen of vancomycin or cefazolin, plus a third- or fourth-generation cephalosporin or aminoglycoside, is anaerobic gram-negative and gram-positive coverage. Consequently, when visceral injury is suspected, the addition of intravenous metronidazole, piperacillin/tazobactam or other antianaerobic antibiotic is mandatory. This suspicion generally arises because of clinical presentation, the results of dialysate cultures, or failure to improve in a timely fashion. Clindamycin is another antibiotic classically used for its anaerobic properties, but clinicians considering its use in this setting should be aware of growing resistance to it by *Bacteroides fragilis* among other organisms.[43–45]

The nephrologist's responsibility does not end, however, with the appropriate antibiotic prescription or even with the request for a surgical consultation. The patient's clinical progress requires close observation. Persistence of moderate to severe peritoneal inflammation, positive effluent cultures, or failure of peritoneal cell counts to dramatically improve within 48 to 72 hours should prompt strong suspicion of either catheter or tunnel infection or undiagnosed visceral injury. The distinction, of course, is critical, because simple catheter removal in addition to an appropriate antibiotic regimen is sufficient in almost all cases of the former,[46] whereas intra-abdominal exploration with intervention appropriate to the specific diagnosis is required in the latter case. As discussed earlier, the presence of "enteric" organisms supports, but does not prove, the presence of visceral injury. Even experienced surgeons may have little experience with PD patients and be unfamiliar with the published experience relating to the assessment of the patients for abdominal catastrophe and the poor outcome associated with specific types of visceral injury in PD patients. Furthermore, they may be understandably hesitant at the idea of laparotomy or even laparoscopy in an acutely ill PD patient with multiple cardiovascular risk factors, peritoneal effluent cultures that may have become negative during antibiotic therapy, and a negative or nonspecific abdominal computed tomography scan. The authors are aware of cases of ruptured appendicitis, perforated sigmoid colon, or gangrenous cholecystitis in whom definitive surgery was delayed from 3 days to 3 weeks in these circumstances. After obtaining the appropriate diagnostic studies, it is critical that a nephrologist who suspects an abdominal catastrophe carefully review the data with the consulting surgeon, sharing any pertinent personal experience and literature. Experience shows that patients are best served by this type of joint discussion between nephrologists and surgeons regarding the need for catheter removal and/or surgical exploration in the PD patient with peritonitis. When serious disagreement persists, a surgical second opinion should be strongly considered.

Finally, as overall peritonitis rates decrease and wait times for deceased donor kidney transplants continue to lengthen, patients are remain-

ing on PD for increasingly longer durations. Long duration on PD, along with high or rapid transporter status, is the major risk factor for the catastrophic and generally fatal complication of encapsulating peritoneal sclerosis (EPS).[47–49] The most common event preceding the development EPS is sudden discontinuation of PD. It has been suggested that long-term (more than 5 to 6 years) PD patients continue to flush the peritoneum periodically for 6 to 12 months to reduce the risk of subsequently developing EPS,[50] although the efficacy of this approach has not been tested in a prospective, randomized trial. It has also been suggested that if long-term PD patients survive a catastrophic episode of peritonitis, a new PD catheter should be inserted as soon as possible to enable resumption of peritoneal lavage, although this is often not immediately possible.

References

1. Pierratos A, Amair P, Corey PN, et al: Statistical analysis of the incidence of peritonitis on CAPD. Perit Dial Bull 2:40-44, 1982
2. Prowant B, Nolph KD: Five years' experience with peritonitis in a CAPD program. Perit Dial Bull 2:169-170, 1982
3. Piraino B, Bailie GR, Bernardini J, et al: Peritoneal dialysis-related infections recommendations: 2005 update. Perit Dial Int 25:107-131, 2005
4. Kim DK, Yoo T-H, Ryu D-R, et al: Changes in causative organisms and their antimicrobial susceptibilities in CAPD peritonitis: A single center's experience over one decade. Perit Dial Int 24:424-432, 2004
5. Li PK-T, Law MC, Chow KM, et al: Comparison of clinical outcome and ease of handling in two double-bag systems in continuous ambulatory peritoneal dialysis: A prospective, randomized, controlled, multicenter study. Am J Kidney Dis 40:373-380, 2002
6. Burkart JM: Comparison of peritonitis rates using standard spike versus Y sets in CAPD. Am Soc Artif Int Organs 34:433-436, 1988
7. Daly CD, Campbell MK, MacLeod AM, et al: Do the Y-set and double-bag systems reduce the incidence of CAPD peritonitis? A systematic review of randomized controlled trials. Nephrol Dial Transplant 16:341-347, 2001
8. Lim CT-S, Wong K-S, Foo MW-Y: The impact of topical mupirocin on peritoneal dialysis infection rates in Singapore General Hospital. Nephrol Dial Transplant 20:1702-1706, 2005
9. Thodis E, Passadakis P, Panagoutsos S, et al: The effectiveness of mupirocin preventing *Staphylococcus aureus* in catheter-related infections in peritoneal dialysis. Adv Perit Dial 16:257-261, 2000
10. Piraino B, Bernardini J, Florio T, et al: *Staphylococcus aureus* prophylaxis and trends in gram-negative infections in peritoneal dialysis patients. Perit Dial Int 23:456-459, 2003
11. Bernardini J, Bender F, Florio T, et al: Randomized, double-blind trial of antibiotic exit site cream for prevention of exit site infection in peritoneal dialysis patients. J Am Soc Nephrol 16:539-545, 2005
12. Fried LF, Bernardini J, Johnston JR, et al: Peritonitis influences mortality in peritoneal dialysis patients. J Am Soc Nephrol 7:2176-2182, 1996
13. Perez Fontan M, Rodriguez-Carmona A, Garcia-Naveiro, et al: Peritonitis-related mortality in patients undergoing chronic peritoneal dialysis. Perit Dial Int 25:274-284, 2005
14. Bunke CM, Brier ME, Golper TA: Outcomes of single organism peritonitis in peritoneal dialysis: Gram negatives versus gram positives in the Network 9 Peritonitis Study. Kidney Int 52:524-529, 1997
15. Szeto CC, Leung CB, Chow KM, et al: Change in bacterial aetiology of peritoneal dialysis-related peritonitis over 10 years: Experience from a centre in South-East Asia. Clin Microbiol Infect 11:837-839, 2005
16. Kern EO, Newman LN, Cacho CP, et al: Abdominal catastrophe revisited: the risk and outcome of enteric peritoneal contamination. Perit Dial Int 22:323-334, 2002
17. Newman LN, Cacho CP, Schulak, et al: Abdominal catastrophe: definition and proposal for a new approach. Adv Perit Dial 17:93-97, 2001
18. Harwell CM, Newman LN, Cacho CP, et al: Abdominal catastrophe: Visceral injury as a cause of peritonitis in patients treated by peritoneal dialysis. Perit Dial Int 17:586-594, 1997
19. Salzer W: Antimicrobial-resistant gram-positive bacteria in PD peritonitis and the newer antibiotics used to treat them. Perit Dial Int 25:313-319, 2005
20. Silva MM, Pecoits-Filho R, Rocha CS, et al: The recommendations from the International Society for Peritoneal Dialysis for Peritonitis Treatment: a single-center historical comparison. Adv Perit Dial 20:74-77, 2004
21. Lye W-C: Empirical treatment of CAPD peritonitis: To each his own? Perit Dial Int 24:416-418, 2004
22. Caring for Australians with Renal Impairment: Evidence for peritonitis treatment and prophylaxis: Treatment of peritoneal dialysis-associated peritonitis in adults. Nephrology 9:S91-S106, 2004
23. Caring for Australians with Renal Impairment: Evidence for peritonitis treatment and prophylaxis: Peritoneal dialysis catheter-related infection: exit site and tunnel. Nephrology 9:S82-S85, 2004
24. Caring for Australians with Renal Impairment: Evidence for peritonitis treatment and prophylaxis: Treatment of peritoneal dialysis-associated fungal peritonitis. Nephrology 9:S78-S81, 2004
25. Van Biesen W, Veys N, Vanholder, et al: Peritoneal-dialysis-related peritonitis: The art of rope-dancing. Nephrol Dial Transplant 17:1878-1882, 2002
26. Digenis GE, Abraham G, Savin E, et al: Peritonitis-related deaths in continuous ambulatory peritoneal dialysis (CAPD) patients. Perit Dial Int 10:45-47, 1990
27. Bunke M, Brier ME, Golper TA: *Pseudomonas* peritonitis in peritoneal dialysis patients: The Network #9 Peritonitis Study. Am J Kidney Dis 25:769-774, 1995

28. Szeto C-C, Chow K-M, Wong TY-H, et al: Conservative management of polymicrobial peritonitis complicating peritoneal dialysis—A series of 140 consecutive cases. Am J Med 113:728-733, 2002
29. Holley JL, Bernardini J, Piraino B: Polymicrobial peritonitis in patients on continuous peritoneal dialysis. Am J Kidney Dis 19:162-166, 1992
30. Kiernan L, Finkelstein FO, Kliger AS, et al: Outcome of polymicrobial peritonitis in continuous ambulatory peritoneal dialysis patients. Am J Kidney Dis 25:461-464, 1995
31. Kim GC, Korbet SM: Polymicrobial peritonitis in continuous ambulatory peritoneal dialysis patients. Am J Kidney Dis 36:1000-1008, 2000
32. Van der Reijden HJ, Struijk DG, van Ketel RJ, et al: Fecal peritonitis in patients on continuous ambulatory peritoneal dialysis, an end-point in CAPD? Adv Perit Dial 4:198-206, 1988
33. Suh H, Wadhwa NK, Cabralda, et al: Endogenous peritonitis and related outcome in peritoneal dialysis patients. Adv Perit Dial 12:192-195, 1996
34. Slingeneyer A, Mion C, Beraud JJ, et al: Peritonitis, a frequently lethal complication of intermittent and continuous ambulatory peritoneal dialysis. Proc Euro Dial Transplant Assn 18:212-221, 1981
35. Wellington JL, Rody K: Acute abdominal emergencies in patients on long-term ambulatory peritoneal dialysis. Can J Surg 36:522-524, 1993
36. Wakeen MJ, Zimmerman SW, Bidwell D: Viscus perforation in peritoneal dialysis patients: Diagnosis and outcome. Perit Dial Int 14:371-377, 1994
37. Asif A, Byers P, Vieira CF, et al: Peritoneoscopic placement of peritoneal dialysis catheter and bowel perforation: Experience of an interventional nephrology program. Am J Kidney Dis 42:1270-1274, 2003
38. Kiefer T, Schenk U, Weber J, et al: Incidence and significance of pneumoperitoneum in continuous ambulatory peritoneal Dial. Am J Kidney Dis 22:30-35, 1993
39. Chang JJ, Yeun JY, Hasbargen JA: Pneumoperitoneum in peritoneal dialysis patients. Am J Kidney Dis 25:297-301, 1995
40. Suresh KR, Port FK: Air under the diaphragm in patients undergoing continuous ambulatory peritoneal dialysis (CAPD). Perit Dial Int 9:309-311, 1989
41. Burkart J, Haigler S, Caruana R, et al: Usefulness of peritoneal fluid amylase levels in the differential diagnosis of peritonitis in peritoneal dialysis patients. J Am Soc Nephrol 1:1186-1190, 1991
42. Caruana RJ, Burkart J, Segraves D, et al: Serum and peritoneal fluid amylase levels in CAPD. Normal values and clinical usefulness. Am J Nephrol 7:169-172, 1987
43. Oteo J, Aracil B, Alos JI, et al: High prevalence of resistance to clindamycin in *Bacteroides fragilis* group isolates. J Antimicrob Chemotherapy 45:691-693, 2000
44. Labbe AC, Bourgault AM, Vincelette J, et al: Trends in antimicrobial resistance among clinical isolates of the *Bacteroides fragilis* group from 1992 to 1997 in Montreal, Canada. Antimicrob Agents Chemother 43: 2517-2519, 1999
45. Dalmau D, Cayouette M, Lamothe F, et al: Clindamycin resistance in the *Bacteroides fragilis* group: Association with hospital-acquired infections. Clin Infect Dis 24:874-877, 1997
46. Choi P, Nemati E, Banerjee A, et al: Peritoneal dialysis catheter removal for acute peritonitis: A retrospective analysis of factors associated with catheter removal and prolonged postoperative hospitalization. Am J Kidney Dis 43:103-111, 2004
47. Jenkins SB, Leng BL, Shortland JR, et al: Sclerosing encapsulating peritonitis: A case series from a single U.K. center during a 10-year period. Adv Perit Dial 17:191-195, 2001
48. Lee HY, Kim BS, Choi HY, et al: Sclerosing encapsulating peritonitis as a complication of long-term continuous ambulatory peritoneal dialysis in Korea. Nephrology 8:S33-S39, 2003
49. Summers AM, Clancy MJ, Syed F, et al: Single-center experience of encapsulating peritoneal sclerosis in patients on peritoneal dialysis for end-stage renal failure. Kidney Int 68:2381-2388, 2005
50. Nakayama M, Yamamoto H, Ikeda M, et al: Risk factors and preventive measures for encapsulating peritoneal sclerosis—Jikei experience 2002. Adv Peritoneal Dial 18:144-148, 2002
51. Prasad N, Gupta A, Sharma RK, et al: Outcome of Gram-positive and gram-negative peritonitis in patients on continuous ambulatory peritoneal dialysis: A single center experience. Perit Dial Int 23:S144-S147, 2003 (suppl 2)