Chapter 19
Peritonitis

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Peritonitis remains a major complication of peritoneal dialysis, accounting for much of the morbidity associated with the technique. Peritonitis accounts for 15–35% of hospital admissions and is the major cause of transfer to hemodialysis (technique failure) [1–7]. High peritonitis rates are associated with mortality, either as a primary or contributing factor [8–11].

In the early 1980s, peritonitis incidence was high, with rates as high as 6.3 episodes/patient year [12]. With improvements in connection technology and institution of prophylactic measures, the rates declined to less than 0.5 episodes/patient year (Fig. 19.1) [4, 13–15]. However, many patients continue to experience frequent peritonitis. There is variability in peritonitis rates by both program and by individual patients. The two most common ways of expressing overall rates are the average number of episodes per single patient year and the average number of months between episodes (Table 19.1). Overall rates mask different outcomes based on patient demographics and the organism involved [2, 16–18]. In small programs, overall peritonitis rates can be skewed by a few individuals with high rates. In these cases, median rates can be useful (Table 19.1) [17]. In larger programs with low peritonitis rates, low overall rates can mask the cases of individuals with high rates. Examining both overall unit rates and individual peritonitis rates is important. This chapter will review the pathogenesis, diagnosis, treatment, and clinical course of peritoneal dialysis-associated peritonitis. Table 19.2 shows the definition of terms used in the chapter.

Pathogenesis

Pathogens

The most common organisms producing peritonitis are summarized in Table 19.3. Most episodes are due to a single organism [19, 20]. In contrast to surgical peritonitis and spontaneous bacterial peritonitis, the most common organisms are Gram-positive [20]. Table 19.3 shows rates (versus percentages) to highlight that the wide variability in reported rates is mainly due to differences in the rate of coagulase-negative staphylococcal peritonitis. With declining coagulase negative *Staphylococcus* rates secondary to changes in connection technique and *Staphylococcus aureus* due to exit site prophylaxis [21] (Fig. 19.2a), the proportion of infections secondary to Gram-negative organisms is increasing [22]. However, the actual rate per year of Gram-negative peritonitis is relatively constant [15, 23, 24] (Fig. 19.2b). Although unusual, fungi are important causes of peritonitis as the sequelae are serious. Fungal peritonitis is predominantly due to *Candida* species, though many species have been reported [25–30]. Anaerobic peritonitis is uncommon and suggests bowel perforation [31–33]. Multiorganism infections that involve more than one Gram-negative organism also suggest bowel perforation. However, polymicrobial peritonitis with Gram-positive organisms can result from touch contamination or a catheter infection [34]. Mycobacterial peritonitis is rare, but may be more common in countries where mycobacterial infections are endemic [35].

Routes of Entry

The routes of entry for peritonitis are summarized in Table 19.4.

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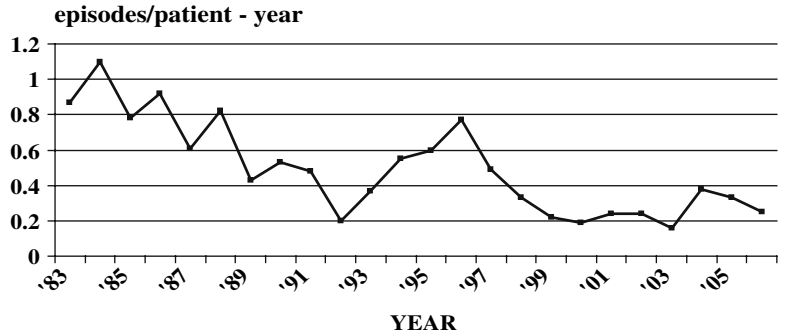

Fig. 19.1 Peritonitis rates over time at the University of Pittsburgh PD Program

Table 19.1 Methods to express peritonitis rates*

1. Rates can be calculated for the program or for an individual patient
a. Number of total peritonitis episodes divided by time period at risk, expressed as episodes/dialysis year
b. Months of peritonitis dialysis at risk, divided by the total number of episodes, expressed as interval in months between episodes
To convert from a or b above to the other, divide 12 by the interval or rate
2. As percentages of patients per period of time who are peritonitis free
3. As median of individual patient rates for the individuals in the program

*Adapted from [145]

Table 19.2 Definitions*

Peritonitis	100 WBC/μL, >50% polymorphonuclear cells
Exit-site infection	Purulent drainage from the exit site. Erythema may or may not indicate infection
Tunnel infection	Erythema, edema, or tenderness over the subcutaneous portion of the catheter (often occult)
Catheter infection	Exit-site and/or tunnel infection
Catheter-related peritonitis	Peritonitis in conjunction with a catheter infection with the same organism or evidence of infection at both sites, though one site may be culture negative
Relapsing peritonitis	Peritonitis with the *same* organism within 4 weeks of stopping antibiotics
Recurrent peritonitis	Peritonitis with a *different* organism within 4 weeks of stopping antibiotics
Refractory peritonitis	Failure of the effluent to clear after 5 days of appropriate antibiotics
Peritonitis related mortality	Mortality secondary to sepsis from peritonitis, with active infection (e.g., positive culture, cell count), during hospitalization for peritonitis or within 14 days of peritonitis episode

*Adapted from [232]

Contamination

The most common source of peritonitis is contamination at the time of the exchange, leading to infection with predominantly Gram-positive skin flora ("touch contamination") [36]. The organism involved is mainly coagulase-negative *Staphylococcus*, though diphtheroids, *Corynebacterium* and *Bacillus* are also seen [37]. The Y-set with flush-before-fill technique decreased the incidence of peritonitis from touch contamination [22, 37–41]. This has decreased coagulase-negative *Staphylococcus* peritonitis as well as other Gram-positives, but has had no effect on the incidence of *S. aureus* [34, 39]. Some patients' skin is colonized with Gram-negatives, which may be related to prior antibiotic use [42]. In these patients touch contamination can lead to Gram-negative peritonitis. In one study the Y-set decreased the incidence of *Acinetobacter* peritonitis, indicating touch contamination as a route of infection [39].

It has been suggested that contamination from mouth organisms (e.g., *Streptococcus* species) can occur in individuals who do not wear a mask during connections, but this is not well studied [43]. Another potential source of peritonitis due to contamination are from bites to the tubing by domestic animals. *Pasteurella* infections have been described most commonly with cats, but there are also reports of peritonitis from hamster tubing bites [44–50].

Table 19.3 Organisms producing peritoneal dialysis peritonitis*

	Episodes/patient-year
Gram-positive	
Staphylococcus epidermidis	0.06–0.4
Stapnylococcus aureus	0–0.15
Streptococcus	0.03–0.14
Enterococcus	0.01–0.04
Other Gram-positive	<0.01–0.02
Gram-negative	0.09–0.24
Pseudomonas aeruginosa	0.01–0.18
Other pseudomonas	0.01–0.02
Klebsiella	0.01–0.02
Escherichia coli	0.01–0.04
Other Gram negative (e.g., *Pasteurella, Morganella, Citrobacter, Acinetobacter, Proteus, Serratia, Enterobacter, Streptophonomonas*)	Each individually <0.01
Polymicrobial with at least one Gram negative	0.02–0.04
Fungal	<0.01–0.07
Mycobacterial	<0.01
Sterile	0.06–0.20

*Adapted from [11, 13, 15, 24, 37, 42, 53, 189, 231, 234, 240, 323–328]

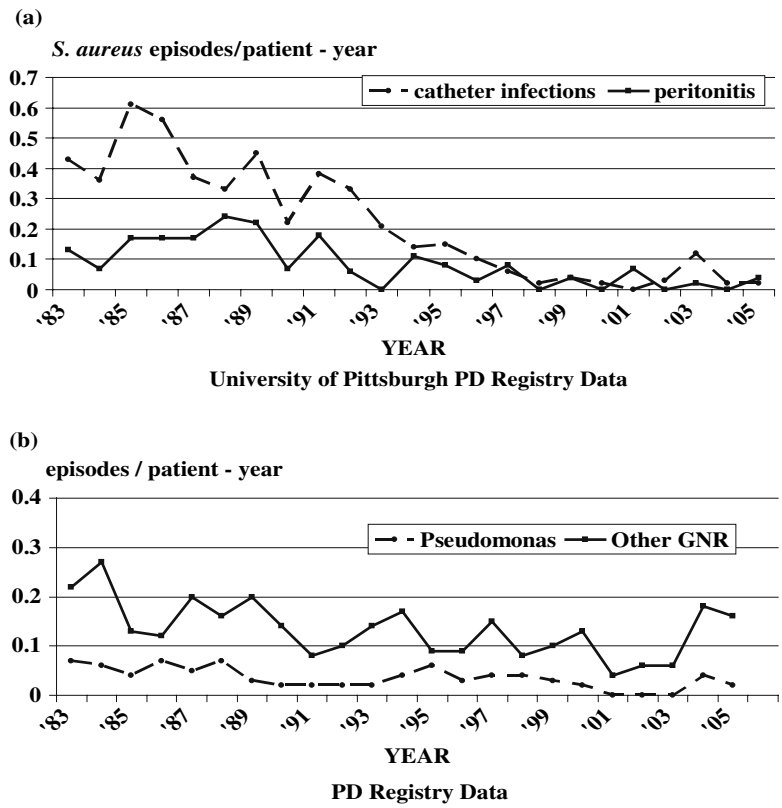

Fig. 19.2 Organism-specific rates over time at the University of Pittsburgh PD Progam. (**a**): *Staphylococcus aureus* catheter infections and peritonitis, (**b**): *Pseudomonas aeruginosa* and other Gram-negative peritonitis

Table 19.4 Routes of entry for peritonitis

Contamination
Catheter-related
Enteric
Hematogenous
Gynecological

Catheter-Related

Catheter infections previously accounted for 10–25% of peritonitis episodes prior to implementation of exit site antibiotic prophylaxis [19, 51–53]. Exit-site and tunnel infections predispose patients to the development of peritonitis, presumably through contiguous spread along the catheter surface [54]. In a trial examining the risk factors for peritonitis, the development of an exit-site infection doubled the risk of subsequent peritonitis [22]. Prior to the introduction of the Y-set, 64% of those with a history of an exit-site infection developed peritonitis, versus 45% without [55]. The most common organisms causing exit-site infections are *S. aureus, Pseudomonas*, and coagulase-negative *Staphylococcus* [56]. Tunnel infections are predominantly caused by *S. aureus*, followed by *Pseudomonas* [52]. In contrast, coagulase-negative *Staphylococcus* is an unusual cause of tunnel infection, catheter-related peritonitis or catheter loss [52, 56]. In a study evaluating catheter-related infections, none of the coagulase-negative *Staphylococcus* peritonitis episodes associated with an exit-site infection required catheter removal versus 76% with other organisms [52] (Fig. 19.3). Peritonitis associated with tunnel infections is generally refractory to treatment without removal of the catheter. Except in the case of coagulase-negative *Staphylococcus*, the treatment failure rate was still high even when there was an exit-site infection without clinical evidence of a tunnel infection, suggesting occult tunnel infections. Catheter infections can also produce relapsing peritonitis, recurrent peritonitis with the same organism within 2–4 weeks of stopping antibiotics. This can be due to the presence of a tunnel infection or alternatively to bacterial colonization of a biofilm [57, 58]. Biofilm formation is ubiquitous and does not necessarily result from infection [59]. Recurrent peritonitis in association with a biofilm is most often due to coagulase-negative *Staphylococcus*, while recurrent peritonitis due to a tunnel infection is generally due to *S. aureus* or *Pseudomonas aeruginosa* [60].

Enteric

Gram-negative peritonitis is caused by intestinal flora but the path of entry into the peritoneal space is not always obvious. Infection can result from abdominal perforation, instrumentation, or other abdominal processes [33, 61–68]. However, in many cases an etiology of the infection is not found [69]. A recent study indicating that Gram-negative peritonitis is reduced with use of gentamicin prophylaxis for routine exit site care is suggestive that some Gram-negative peritonitis occurs via the exit site entrance [21]. It is also quite possible that some peritonitis due to enteric organisms is from touch contamination, as skin flora can contain Gram-negative organisms [70, 71]. Enteric organisms may enter the peritoneal cavity by transmural migration across the gastrointestinal tract without overt gastrointestinal pathology [72]. Diverticulosis appears to increase the risk of transmural migration, as can acute treatment of

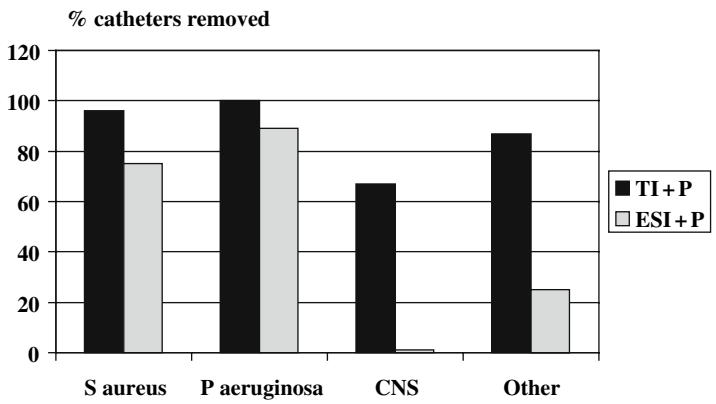

Fig. 19.3 Catheter removal by organism. From Gupta et al., [52]. Reprinted with permission. TI = Tunnel infection, ESI = exit-site infection, P = peritonitis, CNS = coagulase negative *Staphylococcus*

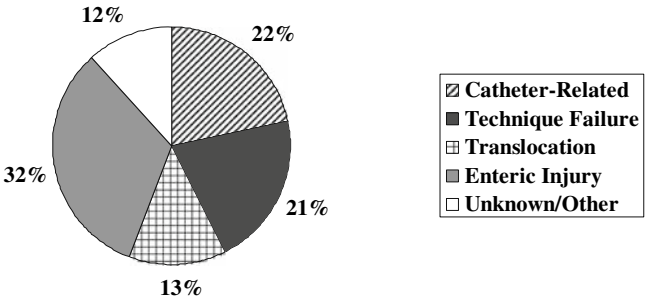

Fig. 19.4 Etiology of peritonitis involving enteric organisms, including *Streptococcus* sp. and *Torulopsis*. Data derived from [67]

constipation [73, 74]. However, the presence of multiple Gram-negative organisms or an anaerobe suggests perforation [31]. Figure 19.4 summarizes the etiology of peritonitis involving enteric organisms.

Hematogenous Spread

Bacteremia can lead to peritonitis, though it is an uncommon cause. Invasive procedures or dental work can produce transient bacteriemia and peritonitis [65, 75, 76]. Routine gastrointestinal endoscopy is associated with bacteriemia in 2–6% of procedures, though esophageal dilation and variceal sclerotherapy have a significantly higher frequency [77, 78]. Dental procedures can lead to peritonitis from mouth organisms such as *Streptococcus* sp. [75, 76]. These cases are potentially preventable with prophylactic antibiotics given at the time of invasive procedures, including dental.

Gynecological

In rare cases, ascending infections from uterine and vaginal sources can lead to peritonitis. This can lead to infections with vaginal flora, including yeast. Cases have been reported with gynecological procedures, vaginal leak of dialysate, and the use of intrauterine devices [79–85].

Predisposing Factors

Risk Factors

Studies examining risk factors for the development of peritonitis identified higher rates for children, African Americans, Native Canadians, and those with a history of substance abuse or lower socioeconomic status [19, 86–91]. The higher rate in children is mainly due to Gram-positive organisms [61]. Age (in adults), diabetes, and gender do not appear to be significant risks, although this is controversial [19, 91–93]. Low serum albumin at the start of dialysis, perhaps signifying inflammation or poor nutrition, has been found to be a risk factor [93, 94]. Immunosuppression is also a risk factor. Prior steroid use is not consistently a predictor, but HIV-positive patients have higher peritonitis rates [19, 95, 96]. In addition, the proportion of Gram-negative and fungal infections is higher in HIV-positive patients [95–97]. Prior antibiotic use is also a risk for fungal peritonitis [28, 30, 98–100]. Gastric acid inhibitors may increase the risk of Gram-negative peritonitis, as can constipation [73, 74]. Upper respiratory tract infections may predispose children to peritonitis, though the reason for this is not clear [76]. The strongest dialysis-related factors are the type of connection system and staphylococcal nasal carriage.

Recent studies suggest that depression may be a risk factor for the development of peritonitis [101]. The reason is not fully delineated but might be due to carelessness in doing the connection by the depressed patient, or alteration of the immune system due to depression, or both. This potential risk factor, and interventions to decrease it, requires further study.

Connection System

The Y-set was introduced in the late 1970s in Europe but did not gain widespread use until the mid- to late 1980s [41, 102]. This system uses a flush-before-fill technique that flushes sterile dialysate into the drain bag after connection to the patient's catheter, but before dialysate is infused into the peritoneum [41, 102]. This decreases the possibility of bacteria from touch contamination reaching the peritoneal cavity. This improvement dramatically decreased the

peritonitis rate when compared to the standard spike system [22, 38, 40, 41]. The Canadian CAPD Clinical Trials Group performed a randomized multicenter trial comparing a standard spike to the Y-set [22]. The Y-set reduced the peritonitis rate by 60%. The twin bag system with a preattached drain bag, requiring only connection at the catheter, further reduces peritonitis rates [103–105]. However, contamination remains a leading cause of peritonitis in many programs and is possibly related to training methods. A structured approach to training is probably best [106]. This requires more formal studies. The very low rates of peritonitis reported from Japan indicate that culture and training may be important in preventing peritonitis [107]. These centers show that rates as low as one episode every 50 months or more are possible.

Continuous cycler peritoneal dialysis is currently a popular form of PD in the Western world. Some studies have found that continuous cycling peritoneal dialysis (CCPD) patients have a lower peritonitis rate than CAPD [108–110] but others have shown this is not the case [111–113]. This is presumably secondary to the decreased number of connections between the system and peritoneal catheter. In the United States some cycler systems require the patient to "spike" bags, which is a potential source of contamination. It is also important not to reuse cassettes or tubing in automatic peritoneal dialysis (APD), as this increases the infection rate [114, 115]. A modification using an assist device to spike the bags on CCPD may further lower peritonitis rates [116]. No controlled studies between CAPD with the twin bag system and CCPD with this modification have been performed.

Staphylococcal Carriage and Exit Site Prophylaxis

Nasal carriage increases the risk of *S. aureus* exit-site infections and subsequently peritonitis [117–121]. Phage typing of *S. aureus* from those with peritonitis or exit-site infection found that in most cases the isolates were the same as from the nares [117, 122, 123]. Zimmerman et al. found that 83% of all *S. aureus* peritonitis episodes were associated with *S. aureus* catheter infection or colonization of the exit site with *S. aureus* [124]. This is consistent with a failure of the Y-set to reduce *S. aureus* peritonitis.

Compared to noncarriers, carriers have a 2–6-fold higher incidence of *S. aureus* peritonitis [15, 117, 121, 125]. Immunosuppressed patients appear to be at particular risk of *S. aureus* peritonitis, regardless of carriage status [126]. Diabetics appear to have an increased rate of nasal carriage [118, 126]. However, it is controversial whether diabetics have an increased risk of *S. aureus* peritonitis after accounting for the higher carriage [118, 125–127]. Treatment strategies that treat nasal carriage (nasal mupirocin or cyclical rifampin) or treat the exit-site to prevent *S. aureus* exit-site infection (mupirocin or gentamicin), dramatically reduce the incidence of *S. aureus* peritonitis (see below, Prevention) [21, 128–132]. Gentamicin compared to mupirocin, both applied daily to the exit site as routine care, decreased both exit site infections and peritonitis, and virtually eliminated *P. aeruginosa* as well as *S. aureus* PD-related infections. This approach has not been compared to intranasal application of mupirocin [21].

Clinical Presentation

The usual symptoms are cloudy fluid and abdominal pain (Table 19.5) [20, 42, 76]. The presentation can vary from cloudy fluid with no pain to a severe illness [42, 133]. In children, cloudy fluid is almost universal, though the incidence of abdominal pain may be less than in adults [76]. The initial presentation in children may be fever alone [76]. The abdominal pain is typically generalized and is often associated with rebound. Local abdominal tenderness could indicate a potential intra-abdominal etiology.

Early studies found that 98–100% of cases presented with cloudy fluid [20, 42, 76]. However, 6% presented with abdominal pain, in the absence of cloudy fluid or elevated cell count [134]. Usually, this represents a delay of

Table 19.5 Clinical presentation* (percentages)

Cloudy fluid	98–100
Abdominal pain	67–97
Abdominal tenderness	62–79
Rebound tenderness	35–62
Fever	34–36
Chills	18–23
Nausea	30–35
Vomiting	25–30
Diarrhea	7–15

*Adapted from [20, 42, 76]

Table 19.6 Differential diagnosis of cloudy effluent

Infectious peritonitis, including culture-negative peritonitis
Eosinophilic peritonitis
Encapsulating peritoneal sclerosis (EPS)
Chylous ascites
Malignant ascites
Pancreatitis
Chemical peritonitis

leukocytosis, and when re-examined, the dialysate cell count has increased [134, 135]. This delay may be secondary to slower cytokine response to infection [134]; therefore, PD patients with abdominal pain should be considered to have peritonitis until proven otherwise. Cloudy fluid can, in rare cases, represent malignancy or chylous ascites [134, 135]. These cases can be differentiated by cytology and dialysate triglyceride levels. The differential diagnosis of cloudy effluent is outlined in Table 19.6.

The presentation is somewhat dependent on the organism involved and the etiology of peritonitis. Episodes due to coagulase-negative *Staphylococcus* and other skin organisms such as *Corynebacterium* are generally milder than episodes with *S. aureus, Streptococcus*, fungi, or Gram-negative organisms [133, 136, 137]. Peritonitis from bowel perforation or other abdominal processes often produces severe symptoms, but the initial presentation may not differ from typical peritonitis [31–33, 138]. Some investigators have found that the presence of free air associated with peritonitis, or alternatively, increasing free air (as free air results from introduction of air during an exchange) to be a clue to bowel perforation [138, 139]. Tunnel tenderness indicates a tunnel infection, but the sensitivity of the physical examination for tunnel infections is low [140]. Since tunnel infections are generally associated with exit-site infections, the presence of an exit-site infection in a patient with peritonitis should trigger a suspicion of catheter-related peritonitis.

Diagnosis

Cell Count

The usual criteria for peritonitis are 1) cloudy fluid; 2) dialysate white blood cell count >100/μL; 3) polymorphonuclear cells (PMN) >50%; and(4) positive culture [20, 26]. The culture is not always positive and is dependent on the technique used for culturing the effluent (see below). Most but not all patients have abdominal pain. In the absence of peritonitis the cell count is usually <30/μL and is predominantly mononuclear [134, 141, 142]. Some authors have found that PMN >50% is a better criterion that the total cell count, especially in patients already on antibiotics [141, 143, 144]. Short dwell times can also decrease the number of white cells seen and in this case, PMN >50% may also be a better criterion [145]. Antonsen et al. found that if the cell count is not done within 4–6 h of collection the number of leukocytes can decrease by 25–30% [146]. The leukocyte count is more stable if samples are sent in EDTA tubes. A number of studies have described the use of leukocyte esterase reagent strips to rapidly diagnosis and elevated leukocyte count in peritoneal fluid [147–150]. This may be useful for units far from a hospital or laboratory.

Tuberculous peritonitis may present with a predominance of lymphocytes, but neutrophil predominance is more common [35, 151–155]. Occasionally, eosinophil predominance is seen in the effluent (eosinophilic peritonitis). Rarely, this is due to fungal peritonitis [156, 157], but in most cases cultures for bacteria and fungus are negative. However, a recent report found that in approximately half of the cases, the eosinophilia was due to infection with a spectrum of organisms similar to the overall distribution in the unit [158]. In many cases the peritoneal eosinophilia occurs early after the initiation of PD and is felt to represent a reaction to the plasticizers in the PD catheter or plastic dialysate bags [159, 160] or inadvertent entrance of air at the time of the exchange [161]. Icodextrin has also been associated with peritoneal eosinophilia [162, 163]. Blood eosinophilia may also be seen in setting of peritoneal eosinophilia [164–166]. In most cases the eosinophils resolve without treatment [159, 164, 167]. Persistent cases may respond to steroids or a mast-cell-stabilizing antihistamine [168–171].

Culture

The handling of the dialysate is important in establishing the etiological agent. Culturing a large volume improves the diagnosis [172]. Blood culture techniques improve the yield of culture and our the standard technique [145, 173–176]. In general, at least 10–20 mL of dialysate should be cultured using blood culture media. Culturing the sediment after

centrifuging 50mL of dialysate is ideal and further decreases the proportion of culture-negative peritonitis [145]. Blood cultures are rarely positive; therefore, routine culturing of the blood is not necessary unless the patient presents with a septic picture. Cultures are generally positive within 24–72 h, though coagulase-negative *Staphylococcus* may grow more slowly [177]. Fungal cultures might take longer than the routine time in many laboratories and a high index of suspicion is needed, especially if the patient is not responding to antibiotics. The growth of mycobacteria is slow, resulting in a delayed diagnosis. Peritonitis due to mycobacteria may be difficult to diagnose but should be suspected if routine cultures are negative, the peritonitis fails to resolve with the usual antibiotics, the patient seems to be chronically ill, and if there is evidence of mycobacterial infection elsewhere. In some cases laparoscopy with biopsy is needed to make a diagnosis. Polymerase chain reaction for tuberculosis can also aid in the diagnosis [178–180].

The use of Gram stain is predominately useful for an early diagnosis of fungal peritonitis, but is much less useful to diagnose bacterial peritonitis [145]. It is important not to base antibiotic therapy solely on the Gram stain. In many cases where Gram-positive cocci were seen, another organism was found, or the culture was negative [181]. However, Gram stains can yield an early diagnosis of fungal peritonitis, which can allow prompt initiation of appropriate treatment and catheter removal [29].

The exit site and tunnel should always be carefully examined in a patient presenting with peritonitis. If drainage from the exit site is present, this should be cultured. Clinical examination of the tunnel often underestimates the presence of a co-existent tunnel infection when exit site infection is present [140]. Ultrasound may be beneficial in diagnosing an occult tunnel infection. The width of a normal tunnel is approximately 6 mm [182]. In the presence of a tunnel infection, ultrasound of the tunnel can show decreased echogenicity around the tunnel, indicating a fluid collection [140, 183]. In patients with an exit-site infection a positive ultrasound indicates a high risk of catheter loss [184, 185]. However, the accuracy of ultrasonography is operator dependent and the indications for use of ultrasound in evaluating the patient with peritonitis have not yet been determined. Vychytil et al. proposed that the indications for tunnel ultrasound in the setting of peritonitis are peritonitis in patient with an exit site infection, infection with *Staphylococcus aureus*, and relapsing/persistent peritonitis [186].

Differential of Culture-Negative Peritonitis

The incidence of culture-negative peritonitis has decreased with improvement in culture techniques. The culture negative rate should be less than 20%; higher rates suggest issues with culture technique [145]. Reculturing sometimes yields an organism [187]. There is debate about the causative organism in these cases, but most studies implicate Gram-positive organisms and the incidence has decreased with the use of the Y-set [37, 188, 189].

Another etiology of culture-negative peritonitis is the use of antibiotics at presentation either surreptitious or for another infection. One study found that in one-third of the culture-negative cases there was antimicrobial activity in the dialysate [190]. Though antibiotic use is a potential cause of negative cultures that should be explored with patients, it is notable that this study had a particularly high rate of culture-negative peritonitis. Culture-negative peritonitis can also be secondary to unusual or difficult-to-culture organisms, such as mycobacteria or some fungi. These cases do not respond to antibiotics, though there may be an early response of mycobacterial infections to quinolones [191].

Pancreatitis can also present as abdominal pain with an increased cell count. Peritoneal fluid amylase >100 U/L can help differentiate pancreatitis from usual peritonitis [192]. However, other abdominal processes, such as ischemic bowel and small bowel perforation, can also produce an elevated amylase in the dialysate [192, 193]. Rare causes of culture-negative peritonitis are chemical peritonitis from medications (Vancoled brand vancomycin, amphotericin, thrombolytics) or presence of endotoxin in the dialysate [194–202]. Icodextrin can also lead to cloudy effluent [163, 203–205]; if this is suspected, stopping the icodextrin will resolve the cloudy effluent.

Treatment

Initial Regimen

Once cultures have been sent, antibiotics should be started promptly. Hospitalization is generally based on severity of illness, such as hypotension, need for intravenous fluids, and parenteral narcotics. Pain control is important and is often neglected [206]. Intraperitoneal antibiotics are generally preferred as this route may be more effective than the intravenous route, and certainly results in high local levels [207]. There are many published antibiotic regimens for PD peritonitis. In an effort to standardize the treatment of PD peritonitis the Advisory Committee on Peritoneal Management of the International Society for Peritoneal Dialysis reviewed the literature and published guidelines. The

guidelines are periodically updated as new information on peritonitis and its treatment becomes available. In 1993, the committee's recommendation for empirical antibiotics was vancomycin plus ceftazidime or an aminoglycoside [208]. However, as concern for vancomycin-resistant organisms increased, the committee updated the recommendations in 1996 to decrease the routine use of vancomycin, and recommended first generation cephalosporins with an aminoglycoside [209]. The most recent update in 2005 recognized that this general recommendation was not adequate for programs with high rates of methicillin-resistant organisms [145]. Methicillin resistance varies from program to program. Therefore, the updated guidelines recommend that the choice of empiric antibiotics be made in light of the patient's and program's history of microorganisms and sensitivities.

Figure 19.5 summarizes the guidelines' initial empirical therapy. The guidelines advocate the use of a drug that covers Gram-positive organisms plus an antibiotic for Gram-negative coverage, including *Pseudomonas*. The actual choice of which antibiotics to use should be based on program organism sensitivity patterns. If there is a high prevalence of methicillin resistance, the recommendation is vancomycin plus a second medication for Gram-negative coverage. The Gram-negative coverage can be obtained with an aminoglycoside, cefepime, aztreonam, or ceftazidime. In general, although both oral and intravenous quinolones have good peritoneal penetration, they do not cover a large proportion of isolates and should not be used for empiric therapy alone, unless local sensitivities support its use. Short courses of intermittent aminoglycosides appear to be safe for empiric antibiotic use, but prolonged or repeated courses should be avoided. If an aminoglycoside is used, it should not be administered in the same exchange as a penicillin, as they are not compatible.

Subsequent antibiotic therapy is based on the culture results (see Table 19.7 for dosing of antibiotics [25, 145, 208, 210]. Both continuous and intermittent therapy dosing are given, and there are not enough data to recommend one regimen over another. Intermittent dosing is more convenient, and may be associated with less toxicity from the aminoglycoside. Once-daily dosing is also applicable to APD, where the antibiotics can be given in the long day dwell (at least 6 h), although few data exist. A randomized multi-center trial in children, many of whom were on cyclers, demonstrated that intermittent administration was as effective as continuous with use of vancomycin or teicoplanin [211]. However, there is concern that intermittent dosing of cephalosporins will lead to periods overnight where the antibiotic level is below the minimal inhibitory concentration (MIC), especially in the presence of significant residual renal function [212]. This theoretically can lead to induction of resistance [213].

Subsequent Regimen

Gram-Positive Organisms

If a Gram-positive organism, especially coagulase-negative *Staphylococcus* or other skin organism, is isolated, the patient should be questioned about a break in technique and a review of connection technique should be made. If *Pasteurella* grows, then the patient should be questioned about cats in the area of the dialysis procedure, as this is often due to cats playing with the tubing. Touch contamination can also lead to polymicrobial Gram-positive peritonitis, which has a better prognosis than polymicrobial Gram-negative peritonitis. The course for various Gram-positive organisms differs and is summarized below.

Coagulase-Negative Staphylococcus and Other Gram-Positive Skin Organisms

If the organism isolated is a diphtheroid, *Corynebacterium* or *Bacillus*, a first-generation cephalosporin for 14 days is generally sufficient. In the case of coagulase-negative *Staphylococcus,* the course depends on whether the organism is methicillin-resistant. Methicillin-sensitive organisms can be treated with cefazolin and the cure rate is equivalent to vancomycin [214]. The cure rate for methicillin-resistant organisms is much lower with cefazolin (versus vancomycin) [214]. If cephalosporins are the empirical therapy the patient should be changed to vancomycin once methicillin-

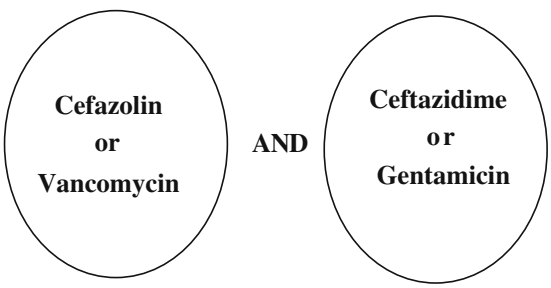

Fig. 19.5 Approach to empiric antibiotic therapy for PD-related peritonitis according to the International Society for Peritoneal Dialysis 2005 guidelines [145]

Table 19.7 Dosing of commonly used intraperitoneal antibiotics for peritonitis*

	Intermittent**	Continuous closing**
Cephalosporins		
Cefazolin	15 mg/kg 20 mg/kg for APD	500 mg load then 125 mg/L
Cephalothin	15 mg/kg/day	500 mg load then 125 mg/L
Cefepime	1 g/day	500 mg load then 125 mg/L
Cephadrine	15 mg/kg/day	500 mg load then 125 mg/L
Ceftazidime	1000–1500 mg/day	250 mg load then 125 mg/L
Ceftizoxime	1000 mg/day	250 mg load then 125 mg/L
Ceftriaxone	1 g/day	250 mg load then 125 mg/L
Aminoglycosides		
Amikacin	2 mg/kg	25 mg load then 12 mg/L
Gentamicin	0.6 mg/kg	8 mg load then 4 mg/L
Netilmicin	0.6 mg/kg	8 mg load then 4 mg/L
Tobramycin	0.6 mg/kg for CAPD For APD 1.5 mg/kg load then 0.5 mg/kg in long dwell	8 mg load then 4 mg/L
Penicillins		
Ampicillin	Data on i.p. not available	125 mg/L
Oxacillin	Data on i.p. not available	125 mg/L
Nafcillin	Data on i.p. not available	125 mg/L
Amoxicillin	Data on i.p. not available	250—500 mg/L load then 50 mg/L
Quinolones		
Ciprofloxacin	Data on i.p. not available (can give p.o. 500 b.i.d.)	50 mg/L load then 25 mg/L
Other antibiotics		
Vancomycin	15–30 mg/kg, up to 2 g/day every 5–7 days	1 g load then 25 mg/L
Clindamycin	Data on i.p. not available	300 mg/L load then 50 mg/L
Imipenem/cilistatin	1 g b.i.d.	500 mg/L load then 200 mg/L
Teicoplanin	15 mg/kg every 5–7 days	20 mg/L
Aztreonam	1,000 mg	1000 mg/L load then 250 mg/L
Ampicillin/sulbactem	2 g every 12 h	1000 mg/L lad then 100 mg/L
Quinupristin/dalfopristin	25 mg/L in alternate bags given in conjunction with 500 mg iv bid	500 mg/l load then 200 mg/L

*Adapted from [25, 145, 208, 210]b
**If significant residual renal function (urine output >100 mL/day), dose should be increased by 25%. i.p., Intraperitoneal; p.o., per os.; APD, automated peritoneal dialysis

resistant organisms are identified. Coagulase-negative staphylococcus can cause biofilm and inadequate antibiotic levels may lead to relapsing peritonitis [215]. The systemic level of vancomycin is generally about twice the level in the effluent and this should be remembered in determining dosing interval. Redosing should occur once the serum level reaches 15 μg/mL to avoid relapse [145, 216].

Staphylococcus aureus

In the majority of cases, *S. aureus* peritonitis is associated with a catheter infection or colonization [122, 124, 137, 217]. The peritonitis tends to be severe and patients often require hospitalization [133, 137]. If *S. aureus* catheter infection is present in conjunction with *S. aureus* peritonitis, the catheter should be removed promptly. Once the culture returns, the subsequent antibiotic regimen also depends on whether the organism is methicillin sensitive. If the organism is methicillin sensitive, the antibiotics can be switched to an anti-staphylococcal penicillin or the first-generation cephalosporin be continued [145]. If vancomycin was used empirically, the antibiotics should be switched to avoid prolonged vancomycin use. Rifampin, for up to 1 week, can be added if desired, or if the response to treatment is slow [145]. If the organism is methicillin resistant, the antibiotics should be changed to vancomycin (or teicoplanin). The vancomycin should be dosed approximately every 5 days with more frequent dosing for those with residual renal function. Trough vancomycin levels can help guide therapy with redosing when the level falls to less than 15 μg/mL [145, 216]. Treatment failure for MRSA is higher than for methicillin-sensitive staphylococcal infections [120]. Antibiotics should be continued for 21 days [145].

Streptococcal

Most cases of Streptococcal peritonitis are secondary to *S. viridans*, followed by *Enterococcus*. Streptococcal peritonitis tends to be more severe with much more pain than episodes due to coagulase-negative *Staphylococcus* [136]. Beta-hemolytic streptococcal peritonitis can be particularly severe, leading to shock and death [42, 218]. Nonenterococcal streptococcal peritonitis responds well to ampicillin and first-generation cephalosporins [145]. The response to these antibiotics appears to be better than the response to vancomycin [145]. Antibiotics should be continued for 14 days [145]. Pain, often severe, must be adequately treated [206].

Enterococcal infections are slower to respond to antibiotics. If sensitive, ampicillin 125 mg/L in each exchange is the preferred antibiotic, to avoid selection of vancomycin-resistant *Enterococcus* (VRE) [145]. Once-daily, low-dose aminoglycosides may be synergistic. *Enterococcus* is part of the gastrointestinal flora; peritonitis should lead to consideration of work-up for abdominal pathology [67, 219]. The incidence of VRE varies from unit to unit, but is a growing problem [220–225]. The prevalence is increased by prior use of antibiotics, especially vancomycin, and hospitalization [221–223]. Linezolid or quinupristin/dalfopristin should be used. Quinupristin/dalfopristin is not effective against *E. faecalis* [145, 224]. Prolonged courses of linezolid can lead to bone marrow suppression and neurotoxicity [145, 224].

Gram-Negative Peritonitis

General Considerations

Once the organisms and the antibiotic sensitivity have been determined, the antibiotics should be adjusted if possible to avoid long-term aminoglycosides, given the risk of ototoxicity and vestibular toxicity [226, 227]. The choice of antibiotics should be based on sensitivities. The aminoglycosides may differ in risk of ototoxicity and vestibular toxicity, and the risk of toxicity may increase with repeated courses [228, 229]. A review in patients without renal failure found that the risk of ototoxicity was 14% for amikacin, 8% for gentamicin, 6% for tobramycin, and 2.5% for netilmicin [229]. The risk for vestibular toxicity was similar for gentamicin, amikacin, and tobramycin at around 3–4%, with netilmicin around 1.5%. However, there are few data for patients on PD. In a study examining the development of ototoxicity using tobramycin, hearing declined in 25% but improved in 17.5% [230].

The most common organisms isolated in non-Pseudomonal Gram-negative peritonitis are *Klebsiella, Escherichia coli*, and *Enterobacter* [69, 231]. The presentation is more severe than that seen with coagulase-negative *Staphylococcus*. Gram-negative peritonitis is associated with higher rates of death, hospitalization, catheter loss, and transfer to hemodialysis than peritonitis with Gram-positive organisms [2, 16, 69, 232]. This is also true for episodes not associated with abdominal perforation.

In uncomplicated episodes the antibiotics should be continued for 2 weeks [145]. Infections with *Acinetobacter* and *Stenotrophomonas* (formerly *Xanthomonas)* can be difficult to treat. *Acinetobacter* is associated with high prevalence of antibiotic resistance and relapse, and is best treated with two antibiotics for 3 weeks [233, 234]. *Stenotrophomonas* also produces serious infections and should be treated with two antibiotics for a duration of 3–4 weeks [145, 235].

Multiple enteric pathogens or the presence of an anaerobe suggest intra-abdominal pathology and the need for surgical evaluation [31, 236]. If a patient with single-organism peritonitis is not responding to appropriate therapy, this should also prompt an evaluation [237]. Patients may initially respond to antibiotics but then deteriorate [67]. Unlike the case with routine peritonitis, bacteremia is not uncommon with peritonitis associated with abdominal processes [68, 238]. In cases of an enteric source of peritonitis (e.g., due to cholecystitis), the recommended antibiotics are metronidazole with ampicillin and ceftazidime or an aminoglycoside [145].

Pseudomonas

Unlike other Gram-negative organisms, *Pseudomonas aeruginosa* peritonitis is commonly associated with catheter infections [239, 240]. Both current and prior episodes of *Pseudomonas aeruginosa* exit-site infection predispose to peritonitis. One study found that 22% of patients with a history of *Pseudomonas* exit-site infection developed peritonitis after resolution of the exit-site infection [241]. If a patient presents with *Pseudomonas* peritonitis, the exit site and tunnel should be examined for infection, which can be subtle. If present, the likelihood of cure without catheter removal is low, and the catheter should be removed [52, 145]. Peritonitis should be treated with two anti-pseudomonal antibiotics [145]. The duration of antibiotics should be 21 days.

Fungal

The optimal treatment of fungal peritonitis is not known. The mortality rate associated with fungal peritonitis is high in children and adults, varying from 20–45% [27, 28, 30, 242–244]. There are reports of successful treatment of fungal

peritonitis without catheter removal, but most patients will ultimately lose their catheter [27, 29, 30]. Larger series have found a cure rate without catheter removal of approximately 10% using fluconazole [245–247]. However, the mortality is high if the catheter remains in place. In the largest reported series, Wang et al. examined the outcome of 70 cases of fungal peritonitis. The treatment regimens varied over time, though most received fluconazole with or without flucytosine [244]. The survival in those whose catheter was removed 71 versus 9% in those whose catheter was not removed, though the series was uncontrolled. The survival was even better if the catheter was removed within 24 h of diagnosis. Similarly, Goldie et al. found that the survival was better if the catheter was removed within 1 week of diagnosis (85 versus 50%) [28]. In that study, the treatment regimen was mainly amphotericin based.

Given the poor outcome, the 2005 guidelines recommend removing the catheter promptly for fungal peritonitis [145]. There are no controlled trials of antifungal therapy. Possible agents are amphotericin, caspofungin, fluconazole, and voriconazole. Amphotericin (0.5 mg/kg/day intravenously ± 1–2 mg/L intraperitoneally) was used in older series, but more recently the azoles have been used [28, 244, 248, 249]. The azoles can be given orally, intravenously or intraperitoneally. However, resistance to azoles has been reported and therefore sensitivities should be checked if possible. 5-Fluorocytosine, where available [50 mg/L intraperitoneally or 1 g per os q.d.), is often added for synergy [142, 214]. If fluorocytosine is used, it is necessary to monitor serum levels to avoid bone marrow toxicity. Therapy should generally be continued after catheter removal for an additional 10 days [145]. Some patients, after catheter removal and treatment, may be able to return to PD but the incidence of adhesions is high and most will need to remain on hemodialysis [29, 244]. The ability to return to PD might be improved by prompt removal of the catheter and antifungal therapy, but this is controversial [244, 250].

Mycobacterium

Mycobacteria are rare causes of peritonitis that require a high index of suspicion for diagnosis. Acid-fast bacilli smears are usually negative, though the ability to detect positive smears can be enhanced by centrifuging 100–150mL of dialysate and examining a smear from the pellet [145]. Cultures, when obtained, are positive, but growth is slow, delaying the diagnosis [35, 154]. The number of reported cases is low, but the disease may respond to standard antimycobacterial therapy [35]. In general, treatment is based on regimens used for extrapulmonary tuberculosis (rifampin, isoniazid, pyrazinamide, ofloxacin plus pyridoxine) [145]. Streptomycin and ethambutol are avoided due to toxicity in end-stage renal disease [145]. CAPD continuation is occasionally possible but ultrafiltration failure may occur [35, 251]. Many patients will have had their catheter removed for unresolving peritonitis before the diagnosis is made. Peritonitis with non-tuberculous mycobacterium, mainly *M. fortuitum*, has been reported and can respond to appropriate antibiotics [252].

Culture-Negative

If, after 96 h, the culture is negative but the patient is responding to therapy (elevated cell count has resolved) and the Gram stain did not reveal a Gram-negative organism, the original antibiotics regimen can be continued, but aminoglycoside should be discontinued. Antibiotics should then be continued for a total of 14 days [145]. If the patient is not responding, the Gram stain and culture should be repeated and special culture techniques should be used for unusual organisms [145, 187]. If this does not reveal an etiology for the apparent failure of antibiotics, the catheter should be removed.

Follow-Up

General

Clinical response is generally seen in 3–5 days, though this is organism-dependent. The dialysate leukocyte count in uncomplicated peritonitis normalizes in 4–5 days [12]. If there has been no improvement by 96 h, re-evaluation is essential. Reculturing might yield an organism not covered by the chosen antibiotics. The patient should be assessed for intra-abdominal pathology or enteric source; the catheter should be evaluated for infection. Unresolving peritonitis predicts a poor outcome and catheter removal is imperative [253, 254].

Catheter Removal

The indications for catheter removal during peritonitis are listed in Table 19.8. Usually, a period of 2–4 weeks between catheter removal and insertion of a new catheter is advocated, to avoid reinfection [145, 255, 256]. However, this requires temporary transfer to hemodialysis, which can be inconvenient for the patient and problematic for young

Table 19.8 Indications for catheter removal during peritonitis

Remove catheter
Refractory
Enteric associated with intra-abdominal process
Fungal
Consider simultaneous replacement of catheter
Relapsing
Catheter-related

children [257]. Experience is growing on simultaneous removal and replacement of catheters for relapsing or recurrent peritonitis [257–265]. To do this safely, the peritoneal WBC count should be less than 100/μL and antibiotics continued for 10 days after the WBC normalized or 7 days after surgery [263]. This simultaneous technique appears to be more successful for Gram-positive peritonitis than for *Pseudomonas* or fungal peritonitis [260, 262]. In one small series a dialysate WBC count <200/μL at the time of the procedure predicted success with *Pseudomonas* infections [259]. In many cases hemodialysis was avoided with the simultaneous procedure [260, 263, 265].

Delayed removal of the PD catheter in refractory peritonitis to 10 days or longer, leads to a very high risk of patient death (approximately 1/3) and to peritoneal membrane failure when the patient attempts return to PD [266] (Fig. 19.6).These results suggest that if the patient is failing to respond to appropriate antibiotics by 5 days, the catheter should be removed more expeditiously [254]. This approach is associated with a considerably lower morbidity.

Relapsing Peritonitis

In relapsing peritonitis, peritonitis with the same organism recurs after completion of antibiotics. Most cases are secondary to the presence of a subcutaneous tunnel infection or involvement of slime layer on the intra-abdominal catheter [58]. In rare cases relapse is secondary to the presence of an abdominal abscess [58]. Relapse may also be secondary to inadequate treatment of the prior infection. Underdosing of antibiotics increases the risk of relapse. Mulhern et al. found that in patients treated with once-weekly vancomycin a low trough level predicted relapse (9/14 with 4-week mean trough <12 mg/L relapsed versus 0/17 > 12 mg/L) [216]. It is important to consider a patient's weight (and hence volume of distribution) and residual renal function when dosing antibiotics. In cases of relapsing peritonitis, the catheter should be removed and as discussed above the catheter can generally be replaced as a simultaneous procedure, allowing the patient to avoid HD or minimize time on HD.

In some cases relapse is secondary to harbouring of bacteria in a catheter biofilm. Once a tunnel infection has been ruled out, the cases may respond to intraperitoneal thrombolytics in addition to antibiotics. Urokinase, streptokinase, and, more recently, tissue plasminogen activator have been used, though urokinase is not currently readily available [267–271]. This treatment is most successful for coagulase-negative *Staphylococcus* or culture-negative peritonitis [271]. The cure rate has been reported to be 50–65% in selected patients, though this is lower than with catheter removal [194, 268–270]. In addition, a recent randomized trial of the use of intraperitoneal urokinase in individuals with peritonitis resistant to empiric antibiotics did not reveal a benefit [272]. If the peritonitis does not respond, the catheter should be removed. This can be replaced as a single procedure if the effluent can first be cleared of white cells.

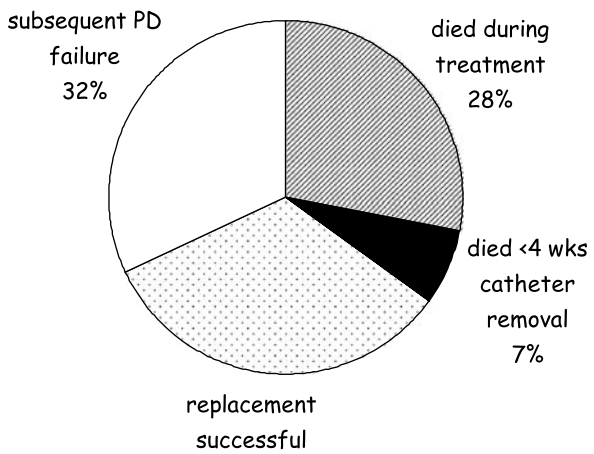

Fig. 19.6 Outcome of peritonitis episodes that fail to respond to antibiotics. Outcomes listed are died during initial therapy before catheter was removed, died within 4 weeks after catheter was removed and before new catheter could be reinserted, attempt at reinsertion of PD catheter unsuccessful and successful reinsertion of PD catheter with resumption of peritoneal dialysis. Data derived from [266]

Outcome and Sequelae

Resolution

From 60 to 90% of episodes resolve with antibiotics [133, 254, 273–275]. The rates of resolution are higher in the absence of an exit-site or tunnel infection. Catheter removal rates are higher for *S. aureus* and Gram-negative infections [52]. The higher rate of catheter removal for *S. aureus* is secondary to catheter infections, as the rate of removal in the absence of a catheter infection is similar to coagulase-negative *Staphylococcus* [69].

Abscess Formation

Abscess formation occurs in less than 1% of episodes of peritonitis [276]. The patients tend to present with abdominal pain, nausea, vomiting, and peripheral leukocytosis [276]. The organisms reported are Gram-negative, fungus, and *S. aureus* [58, 276]. CT scan or ultrasound is helpful in making the diagnosis. The disease responds to drainage.

Transfer to Hemodialysis (Technique Failure)

Peritonitis is a major cause of technique failure in PD patients, accounting for 30–80% of permanent transfers [4, 277, 278]. The data on whether technique failure secondary to peritonitis has declined with the decline in overall peritonitis rates are conflicting [7, 279]. The Y-set reduced peritonitis, but did not significantly impact technique survival in all studies [4, 189]. Peritonitis from coagulase-negative *Staphylococcus* and other skin organisms tends to be less severe, and as a result reduction in touch contamination has less of an impact on technique survival. Severe episodes of peritonitis are associated with decreasing albumin from increased protein losses, poor intake and inflammatory response [18, 280]. This is associated with a worse long-term outcome. Prompt transfer to hemodialysis is appropriate in severe, poorly responding episodes of peritonitis.

Encapsulating Peritoneal Sclerosis

Encapsulating peritoneal sclerosis (EPS) is an uncommon but serious complication of PD. The frequency is variable but tends to increase with longer time on PD [281–284]. This entity is not infectious, but patients present with abdominal pain, nausea and vomiting, bowel obstruction and malnutrition and sometimes low grade fever [281, 285]. A loss of ultrafiltration is seen [281, 285]. The disease can present after transfer to hemodialysis or transplantation as ascites. Recurrent peritonitis may be a predisposing factor, but this is not a consistent finding. Nomoto et al. found that those with EPS had a 3.3 times higher peritonitis rate than those without, but Hendriks et al. found that the rates were not different [285, 286]. In a recent study by Yamamoto et al., the two independent risk factors associated with EPS were membrane transport characteristics and number of peritonitis episodes [284]. The pathogenesis is a fibrotic reaction of the peritoneum. It may not be recurrent peritonitis alone but a severe episode that is important [287], especially after a fairly long period on PD. This is supported by Davies et al., who found that ultrafiltration tends to decline with time on PD and is worsened and accelerated by severe or closely spaced episodes of peritonitis and to use of higher dextrose dialysate [288].

Death

Peritonitis results in death in 1–6% of episodes [9–11, 289]. The immediate cause of death is often cardiovascular [9] and patients with cardiovascular disease appear to be at increased risk of death after peritonitis [18]. The mortality rate for Gram-negative and fungal peritonitis is significantly higher (4–10% for Gram-negative, 20–45% for fungal) [9, 28, 69, 232, 242, 244] (Fig. 19.7). Mortality associated with bowel perforation approaches 50% [67, 68]. In contrast, the mortality associated with coagulase-negative *Staphylococcus* peritonitis is less than 1% [232]. There is a high mortality rate in the first year after transfer to hemodialysis, which may be related to poorer nutrition in those with severe peritonitis episodes [4]. Lower albumin levels are associated with increased mortality after peritonitis [290], but this may be related to the increased protein losses with severe peritonitis and not pre-existing malnutrition. Elevated C-reactive protein levels also predict death after peritonitis [9], so another explanation for the reason lower albumin predicts death is inflammation.

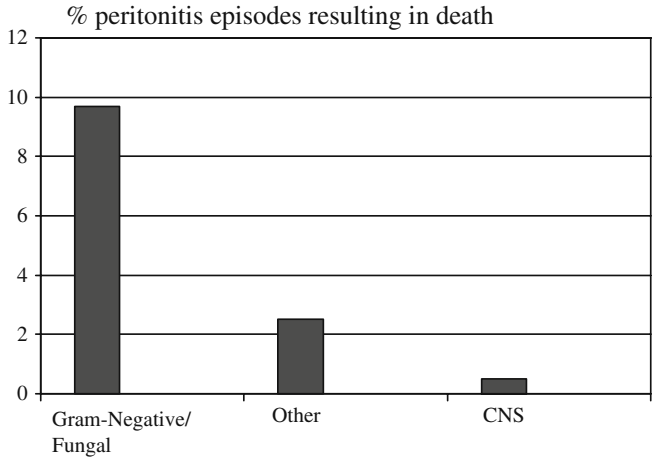

Fig. 19.7 Mortality of peritonitis by organism. Rates expressed as percentage associated with death per episode. CNS = Coagulase-negative *Staphylococcus*. Data derived from [232]

Prevention

General

Training of patients in the PD technique by experienced nurses is critical. Dryden et al. found that a preventative program decreased the risk of exit-site infection (10-fold), peritonitis rate (2-fold), and catheter loss (4.5-fold) [291]. The program was directed at *S. aureus* nasal carriage, intensive training of nurses, and aseptic techniques for catheter insertion and care. A recent study found that a standardized training curriculum reduced exit site infection rate (0.22 versus 0.38 episodes per patient year ($p = 0.003$), with a trend toward lower peritonitis rates (0.33 versus 0.44 episodes per patient year, $p < 0.10$) [106]. In terms of training, patients should be instructed to wear masks during exchanges. Careful hand-washing with antibacterial soap, and complete drying of the hands, decreases the skin bacterial count by 95–99%, thus reducing the potential transfer of bacteria [292]. The room where exchanges are performed should be isolated from heavy traffic. Pets should be excluded from the room in which exchanges are performed as bacterial transmission from pets has been reported [44–50]. The ISPD web site has a video program on teaching nurses how to instruct patients on the proper PD technique, freely available without charge to all at ispd.org.

Tubing changes should be performed by nurses, not patients [293]. Connection technology for CAPD using a twin-bag system or APD should be utilized. In areas where prespiked APD bags are not available, the spike assist device for APD may also decrease rates [116]. Aggressive nutritional intervention in children may decrease the peritonitis rate [294]. We provide prophylaxis for technique-related contaminations with cefazolin or cephalexin, as well as prophylaxis for invasive procedures [295].

Prevention of Catheter-Related Peritonitis

The use of prophylactic antibiotics at the time of insertion is recommended by the ISPD guidelines [145]. There have been four randomized prospective trials. Gadallah et al. randomized catheter procedures into three groups: vancomycin (1 g intravenously approximately 12 h before catheter placement, $n = 86$), cefazolin (1 g approximately 3 h before catheter placement, $n = 85$) or no antibiotics ($n = 83$) [296]. One patient developed peritonitis in the vancomycin group within 14 days of the procedure versus 6 in the cefazolin group and 10 in the group who did not receive antibiotics. This suggests that vancomycin may be more effective than cefazolin. Two other studies showed a benefit of preoperative antibiotics using cefuroxime (1.5 g intravenously, 250 mg intraperitoneally) or gentamicin (1.5 mg/kg intravenously) [297, 298]. In both cases the incidence of peritonitis was lower in the first month after insertion. In contrast, Lye et al. found no benefit using gentamicin (80 mg) and cefazolin (500 mg) [299].

No particular catheter has been definitively shown to have lower infection rates than the standard silicon Tenckhoff catheter. Double-cuff catheters should be utilized, with a downward or lateral directed exit site [19, 256, 300]. Swimming should be avoided after catheter insertion, until the catheter is healed, and swimming in lakes and ponds completely avoided. Exit-site infections should be treated promptly. We have found that patients with untreated well water at home are at increased risk for *Pseudomonas* exit-site infections, and we instruct these patients to use bottled water for exit-site care.

Specific Organism Prophylaxis

Dental Procedures

Oral procedures have a high incidence of transient bacteremia, though the inoculum is generally low [77]. Peritonitis after dental procedures has been reported in children and adults [75, 76]. Though there are no prospective trials, we would recommend prophylaxis prior to dental procedures, using American Heart Association guidelines [295].

Staphylococcus aureus

S. aureus nasal carriage increases the risk of peritonitis as well as exit-site and tunnel infections [117–119, 121, 301]. There have been a number of studies examining the effect of prophylaxis on peritonitis; these regimens are summarized in Table 19.9. All the regimens decrease the rate of infection. The results of the randomized trials using mupirocin or rifampin (versus no treatment) in adults are shown in Fig. 19.8. In a study directly comparing exit-site mupirocin to cyclical rifampin every 3 months, both were equally effective but the incidence of side-effects was greater with the rifampin [131]. A recent randomized controlled trial of exit site mupirocin versus exit site gentamicin found similarly low *Staphylococcus aureus* catheter infection rates (0.06 versus 0.08 episodes/pt year for mupirocin versus gentamicin, respectively) [21]. The low rate of *S. aureus* catheter infections was accompanied by a low rate of *S. aureus* peritonitis in both arms as well. The overall peritonitis rate was lower in the gentamicin group compared to the mupirocin group due to a decrement in other organisms (0.34 versus 0.52 episodes/patient year) (Fig. 19.9).

In two studies utilizing mupirocin at the exit site versus no treatment, all patients were treated regardless of nasal carriage status [131, 132]. In contrast, the nasal mupirocin trials treated carriers only [121, 130, 302]. Most of the nasal mupirocin trials required subsequent surveillance cultures and retreatment of those who became recolonized [121, 130]. The recolonization rate is high after mupirocin or rifampin treatment, with 40–55% recurrence at 3 months and 60% at 12 months [130, 303, 304]. The Mupirocin Study Group, in contrast, initially screened patients for nasal carriage (two-thirds positive cultures) and then treated identified carriers with nasal mupirocin for 5 days every month regardless of subsequent nasal culture results [302]. Given the cost of frequent cultures, this may be a more economical approach but did not reduce *S. aureus* peritonitis rates, just exit site infection rates. The definition of carrier varied between the studies, but a conservative definition is one positive of three serial cultures [131]. Persistently positive nasal cultures (two or more out of three) are associated with a greater risk of infection, but the staphylococcal peritonitis rate is still elevated with one positive culture (compared to noncarriers) [119]. Exit site prophylaxis may be preferred as one study found that the strains of S. *aureus* isolated from the exit site may not be the same as the nose, indicating that the nose is not the only source of colonization [122]. Since publication of the studies demonstrating efficacy of mupirocin, a number of studies using historical controls have found that mupirocin is effective at decreasing exit site infection and peritonitis rates [304–307].

Table 19.9 *Staphylococcus aureus* prophylactic regimens

	Reference
Rifampin (carriers)	
Adults:	
600 mg/day × 5 days every 3 months	[129, 131]
Children:	
300 mg/day for children <30 kg, 600 mg/day for >30 kg	[303]*
20 mg/kg/day in two divided doses	[128]**
Exit-site mupirocin	[131, 132]
Daily as part of routine exit-site care for all patients (not studied in children)	
Nasal mupirocin	
2% b.i.d.–t.i.d. × 5–7 days for carriers with retreatment for recolonization based on cultures (adults and children)	[121, 130, 304]
2% b.i.d. for 5 days each month in carriers	[302]
Anti-Pseudomonal antibiotics, covering Staphyloccocus (not studied in children)	
0.1% gentamycin cream daily as part of routine exit-site care for all patients	[21]
0.5 mL (1 mg) ciprofloxacin otologic solution daily as part of routine exit-site care for all patients	[318]

*Pediatric trial; the rifampin was given with nasal bacitracin
**Pediatric trial; the mupirocin was given with cloxacillin

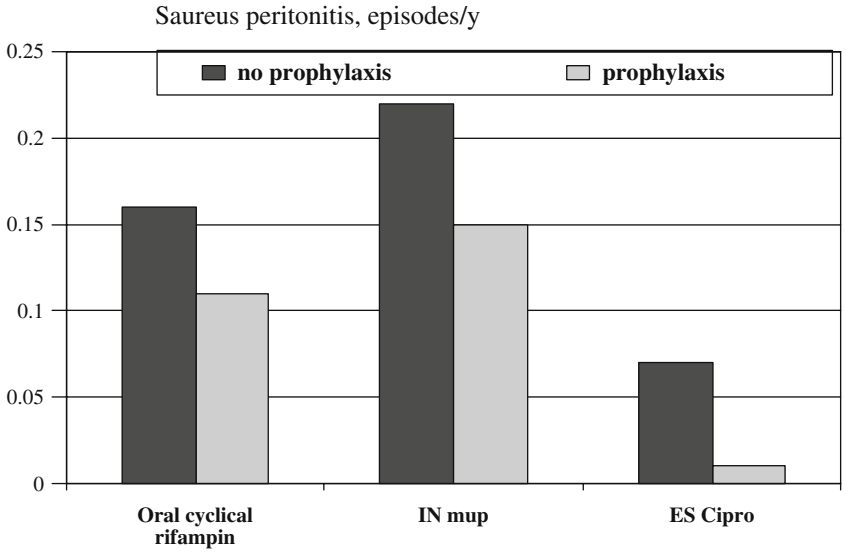

Fig. 19.8 Effect of staphylococcal peritonitis prophylaxis on peritonitis rates. Trials shown are randomized trials in adult trials only rifampin or mupirocin vs. no treatment or placebo. Data derived from [129, 302, 318]. ES mup = Exit-site mupirocin, IN mup = intra-nasal mupirocin

There are few side-effects associated with mupirocin, mainly nasal irritation and discharge for the nasal route [302]. Exit-site mupirocin ointment can degrade polyurethane and should be avoided with these catheters, which are rarely used [308]. Increased antibiotic resistance to mupirocin has been reported, though the prevalence varies among programs. Perez-Fontan found an increasing prevalence of mupirocin resistance over time [309]. In that program, mupirocin treatment was intermittent and based on surveillance cultures. Increased courses of mupirocin were associated with an increased prevalence of resistance. Resistance was not associated with an increased peritonitis rate, but there was an increase in exit site infection, raising the concern that in the future, mupirocin will be less effective. Mupirocin appears to be less effective for MRSA [310, 311].

Given the high morbidity associated with *S. aureus* peritonitis, each dialysis unit should establish a prophylactic regimen to prevent this infection. This might be cyclical intranasal application of mupirocin, daily exit-site mupirocin, or daily exit-site gentamicin. All appear to be effective in reducing *S. aureus* infections in PD patients, and the latter in addition reduces Gram-negative infections.

Fungal

Prior antibiotic use increases the risk of fungal peritonitis [22, 28, 98]. This introduces a potential target group for prophylaxis. Six trials have studied the effect of prophylaxis, though only one was a prospective, randomized trial. Five

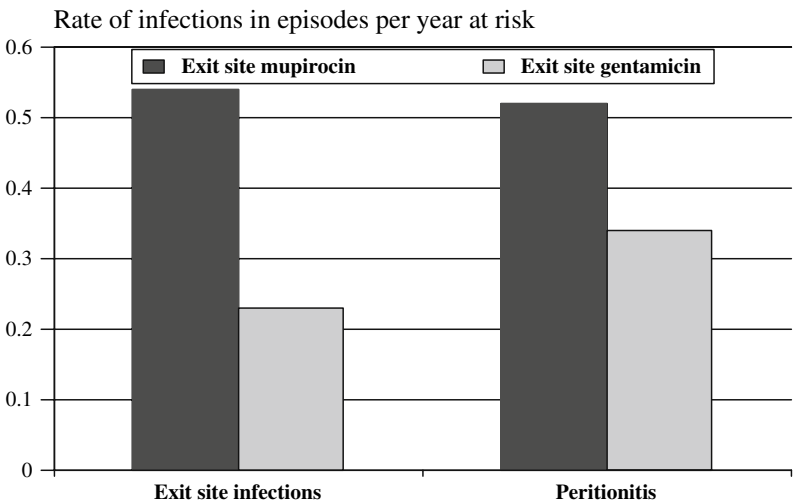

Fig. 19.9 Comparison of exit site infection rate and peritonitis rates with exit site mupirocin vs. exit site gentamicin. Data derived from [21]

trials utilized oral nystatin: 500,000 U tablet q.i.d., 500 IU t.i.d., or in children 10,000 U/kg/day in three divided doses during the antibiotic course [312–316]. Lo et al. randomized patients to either nystatin tablets during antibiotic courses or to control [313]. There was a decreased incidence of *Candida* peritonitis (1.9 versus 6.4 per 100 peritonitis episodes and 0.66 versus 1.43 per 100 antibiotic prescription for any indication) with prophylaxis. Two of the trials utilized ketoconazole (10 mg/kg per day in children) or fluconazole (200 mg on day 1, then 100 mg/day) [315, 317]. These studies, which had retrospective controls, also found a decreased incidence of fungal peritonitis. Two studies using either retrospective controls or compared one dialysis unit utilizing prophylaxis with another that did not, did not find that nystatin was effective [312, 314]. However, both of these units had low baseline fungal peritonitis rates and prophylaxis may be more effective when baseline rates are high. No side-effects of the prophylactic regimens were reported.

Gram-Negative

As most pseudomonal peritonitis is due to catheter infection, prophylaxis is possible. Two studies have investigated exit site antibiotics as a preventive measure to prevent peritonitis. Bernardini et al., randomized 133 individuals to exit site mupirocin or gentamicin cream [21]. The catheter infection rate and the peritonitis rate were lower in the gentamicin group. Of note, there were no pseudomonal exit site infections or peritonitis episodes in the gentamicin group, versus 6 catheter infections (0.11 episodes per patient year) and 2 peritonitis episodes (0.04 episodes per patient year) in the mupirocin arm. Both the pseudomonal and nonpseudomonal Gram-negative peritonitis rates were lower with the gentamicin, suggesting that some Gram-negative infections are related to catheter infections. Montenegro et al. randomized 164 individuals to exit site care with soap and water only versus exit site care with soap and water plus application of 1 mg ciprofloxacin (0.5 mL otologic solution) [318]. Ciprofloxacin reduced overall, Staphylococcal and Pseudomonal exit sites infection rates. Similar to the gentamicin trial, there were no episodes of Pseudomonal infections in the treated group.

Unfortunately, despite the morbidity and mortality associated with non-pseudomonal Gram-negative peritonitis, there are few effective interventions to reduce the incidence. Trials utilizing neomycin, cotrimoxazole, or cephalexin were not effective in decreasing peritonitis [319–321]. Constipation, a possible inciting event, should be avoided with a bowel regimen. Prophylactic antibiotics should be administered for endoscopic and gynecological procedures. The abdomen should be drained prior to procedures. Further research aimed at preventing non-pseudomonal Gram-negative peritonitis is necessary.

Quality Improvement

The ISPD guidelines state that a center's rate should be no more than 0.67 episodes per patient year (1 episode per 18 months) [145]. Each dialysis program should monitor individual patient and overall peritonitis rates. The presumed cause and organism patterns should be evaluated as part of a continuous quality improvement (CQI) program. Interventions directed at the cause of peritonitis should be made to prevent future episodes. CQI can involve training processes, retraining, exit site prophylaxis and treatment of contaminations. Borg et al. found that with a multifaceted quality improvement project, they were able to decrease the peritonitis rate from 1.6 episodes/patient year in 1998 to 0.3 episodes/patient year in 2002 [322]. Individuals with high peritonitis rates can be problematic. Technique should be reviewed and the patient retrained. A careful evaluation for an occult tunnel infection should be performed and consideration made for changing the catheter. If these maneuvers are not effective in decreasing the peritonitis episodes, then one should consider transfer to hemodialysis. With the maneuvers stated above, hopefully centers can achieve very low peritonitis rates and thus decrease the morbidity associated with these infections.

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