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Diagnosis and Treatment of Peritonitis in Patients on Continuous Ambulatory Peritoneal Dialysis

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Abstract:*Peritonitis is frequently associated side effect of peritoneal dialysis of patients on continuous ambulatory peritoneal dialysis (CAPD).Clinical symptoms of CAPD include abdominal pain, tenderness, often rebound, and cloudy dialysate fluid. Common pathogens are 60% to 80% Staphylococcus epidermidis,Staphylococcus aureus, Streptococcus spp.Nasal carriage ofS.Aureus is associated with and increased risk of catheter-exit site infection.Encapsulatingsclerosis peritonitis (ESP) occurs in 1% to 5% of patients. Increased duration of peritoneal dialysis has been assumed to be a risk factor for ESP. Diagnosis is made when microorganisms and increased number of leukocytes, dialysate turbidity in combination of clinical findings Vancomycin, ceftazidime, cefepime, carbapenem and fluoroquinolone are antimicrobials of choice. Prognosis of peritonitis in dialysis patient is favorable. Clinical outcome is often worse in cases of secondary peritonitis. Mortality in 6% of 565 patients with 693 episodes was reported in a retrospective study.*
Keywords*:Continuous ambulatory peritoneal dialysis,Peritonitis, Diagnosis, and Treatment.*

I. Introduction.

Peritonitis is a common complication of peritoneal dialysis. Peritonitis is associated with significant morbidity, catheter loss, transfer to hemodialysis, transient loss of ultrafiltration, possible permanent membrane damage, and occasionally death [1,2].Peritoneal dialysis has been used successfully to treat uremia patients with end stage renal disease since the mid 1940's.Peritonitis was a frequently associated side effect that hindered the acceptance of chronic peritoneal dialysis until an improved access catheter was developed by Henry Tenckhoff in 1968.This catheter significantly decreased the incidence of peritonitis, but initial of patients undergoing continuous ambulatory peritoneal dialysis(CAPD) with this catheter indicated peritonitis rates of more than six episodes per patient per year[3].This rate has appeared to decline with introduction of collapsible plastic bags, improved adapters(Y system) and better techniques[4]. However, peritonitis remains the major complication of CAPD today and is the chief reason for peritoneal catheter loss, discontinuation of peritoneal dialysis, and switch to hemodialysis [5].Historically it occurred at a rate of about one episode per patient per year(range<0.5 to ≥).Of patients,45% experienced peritonitis at least one during their initial 6 months of CAPD treatment. This rate increased to 60% to 70% during the first year [6].During the recent years CAP has become recognized as a major form of therapy for end-stage renal disease. Despite continuous advances, peritonitis remains a major limiting factor in the widespread application of CAPD[6].Frequently isolated organisms include 60 % to 80% *Staphylococcus epidemidis*,,*Staphylococcus aureus*,*Streptococcus*spp,and*diphtheriods*[7].Clinical manifestations include abdominal pain, tenderness (often with rebound) and a cloudy appearance of the drainage dialysate fluid are common)[8]Encapsulating peritoneal sclerosis(ESP) occurs in 1% to 5% of patients [8].Diagnosis can be assumed when dialysate contains more than 100 leukocytes /mm^3[9].Intraperitoneal instillation or intravenous single dose of cefepime and vancomycin antibiotics are used[9].The clinical outcome is often worse in cases of secondary peritonitis[10].Paper reviews the diagnosis and treatment of peritonitis in patients on CAPD.

II. II.Contributory factors

The origin of infection in most cases seems to be contamination of the catheter by common skin organisms [3].Alternations of skin flora in CAPD recipients may lead to peritoneal contamination with enteric pathogens [11].A higher incidence of peritonitis seems to occur in patients undergoing dialysis who are carriers of nasal*S.aureus*. Although this carrier state increases the likelihood of developing exit site infections in comparison with non-carriers, the overall peritonitis rate of two groups was not different in one study, although all cases of *S.aureus* peritonitis occurred in the carriers [12].Pathogens also may contaminate the peritoneum from exist site infections and subcutaneous-tunnel (periluminal) infections, transient bacteremia,

andcombination of the dialysate system during bag exchanges. Enteric bacteria may also gain access to the peritoneal cavity by transmural migration through an intact intestinal wall after the introduction of hypertonic solutions into peritoneum. This mechanism may account for enteric peritonitis in patients undergoing dialysis. One rare occasion, a vaginal leak may serve as a source of peritonitis. Polymicrobial infection with fecal organisms is suggestive of bowel perforation as complication of catheter placement or secondary peritonitis from other causes [12].

Alteration in peritoneal defenses may increase the risk of peritonitis in patients undergoing CAPD.The antimicrobial function of peritoneal macrophages and polymorphonuclear cells generally requires the presence of opsonins.A reduction in the levels of immunoglobulin G and G3 has been noted in peritoneal dialysis effluents in comparison with serum, and the concentrations of these crucial opsonizing agents are related inversely to the frequency of peritonitis [13].Other important factors that impair host defense mechanisms are low pH and high osmolality of peritoneal dialysis fluid,both can impair polymorphonuclear leukocyte function and antibiotic efficacy. Newer peritoneal dialysis fluids containing glucose polymers (e.g.,icodextrin) may be less detrimental to macrophage andpolymorhonuclear leukocyte activity. The formation of biofilm on the catheter appears to contribute to relapsing or recurrent infection, as well as to decrease therapeutic responses and development antimicrobial resistance [13].

Among peritoneal dialysis patients, peritonitis may be peritoneal dialysis related or secondary (such as enteric rarely hematogenous).Peritoneal dialysis-related peritonitis is due to touch contamination with pathogenic skin bacteria or to catheter- related infection. Secondary peritonitis is caused by underlying pathology of the gastrointestinal tract and has rarely been reported due to hematogenous spread, such as post dental procedures. Gastrointestinal conditions that may lead to secondary peritonitis include cholecystitis, appendicitis, ruptured diverticulum, and treatment of severe constipation, perforation during endoscopy, bowel ischemia, and increased hernia. Secondary peritonitis is less common than peritoneal dialysis-related peritonitis. As an example, in one review,intra-abdominal pathology was responsible for less than 6 percent of cases of peritonitis in chronic ambulatory peritoneal dialysis [14].The clinical outcome is much worse in cases of secondary peritonitis. In one study report,11 of 26 patients with secondary peritonitis died[10].Compared with an overall peritonitis-associated mortality of approximately 2 to 3 percent among all peritoneal dialysis patients with peritonitis[15].

III. III. Microbiology

Wittmann and colleagues content that Staphylococcus organisms (coagulase-negative) and *S.aureus* (often methicillin resistant) predominate.*Streptococcus* species are isolated 10% to 15%.Gram negative bacteria or yeast occurs in patients with recurrent episodes. A single organism usually is isolated. Gram stain of the effluent generally are negative, with 9% to 40% are reported in several series. If multiple species or anaerobes are isolated, bowel perforation should be suspected [8].Finkelstein and associates confirmed that gram positive organisms constitutes 60% to 80% of isolates most commonly *S.epidermidis*,followed by *S.aureus*,*Streptococcu*s spp,anddiphtheroids.*Staphylococcal* isolated have been noted to grow on polymer surfaces and frequently produce extracellular slime layer or biofilim that may protect these bacteria from host defenses[7].Biofilms are complex aggregates of extracellular matrix and interdependent microorganisms from multiple species, many may be difficult or impossible to isolate using standard clinical laboratory techniques[16].*Bacteria found in biofilms have their antibiotic resistance increased up to 1000times* when compared to free living bacteria of same species. A recent study found that biofilms were present on mucosa of 75% of patients undergoing surgery for chronic sinusitis [17].The ability of biofilm formation seems to play an essential role in the virulence of coagulase-negative staphylococci (CNS)[18].

Staphylococcus aureus nasal carriage plays an important role in infection in the CAPD patients.Yu and colleagues established that *S.aureus* infection occurred significantly more frequently in nasal carriers of the organisms than the non-carriers [19].Previous investigations among patients undergoing CAPD[20] have suggested a possible link between the carriage of this organism and exit site infection, peritonitis or both. However, the number of patients studied was relatively small, and most were studied after CAPD was started. Luzar and colleagues in a study of 63 patients concluded that in patients beginning ambulatory peritoneal dialysis, the nasal carriage of *S.aureus* is associated with and increased risk of catheter-exit site infection and that the performance of nasal cultures before the implantation of the catheter can identify patients at high risk of subsequent morbidity [21].

The etiology of Sclerosing encapsulating peritonitis or encapsulating peritoneal sclerosis (EPS) secondary to PD include severe and or/ non-resolving peritonitis, especially that due to *Staphylococcus aureus*, fungi, and *Pseudomonas*spp,and especially in the long-term patients increased. Increased duration of PD has been assumed by some to be a risk factor for EPS, but first reported correlation between increased time on dialysis and increased risk of EPS comes from the Australian study. According to Rigby, the prevalence of EPS increased from 1.9% at longer than 2 years, to 19.4% at longer than 8 years on PD [22].

Vancomycin-resistant enterococci as nosocomial pathogen is now more frequently recognized in peritonitis cases. Gram-negative organisms are obtained from 15% to 30% of isolates. *E.coli* is the most common, followed by *Klebsiella* and *Enterobacter*spp., *Proteus* spp., and *Pseudomonas*spp. Cases caused by gram-negative organisms have increased according to a retrospective study of peritoneal dialysis-related peritonitis over decade-long period[23].Less common pathogens include Acinetobacter spp., *Candidaalbicans*, and anaerobic bacteria. Rare isolates include atypical *Mycobacteria(*usually *Mycobacteriumcheolani* or *Mycobacterium fortuitum*), *M.tuberculosis, Candidaparapsilosis, Aspergillusfumigatus, Norcardiaasteroids*, and *Fusarium*spp. Polymicrobial peritonitis in patients undergoing peritoneal dialysis usually is assumed to be secondary to a primary intestinal process (e.g.,bowel perforation) and usually necessitates surgical exploration[24].

IV. Diagnosis

Diagnosis of peritonitis is made when microorganisms and an increased number of leukocytes are present in the dialysate in combination with a constellation of clinical findings that include abdominal pain and tenderness (60% to 80% of patients) nausea and vomiting (3)%),fever (10% to 20%),and diarrhea(10%).Not all these criteria need to meet however, to fill the diagnosis[3].The dialysis almost always is cloudy and microscopic examination reveals a leukocyte count greater than 100 cells/mm^3(in approximately 85% of cases, more than 500/mm^3,with neutrophils predominately. The length of the dwell time has an impact on the number of effluent cells. A low leukocyte count in dialysate may also be indicative of a **tunnel infection** and not peritonitis.Although a predominance of lymphocytes may be encountered with fungal and mycobacterial infection, the majority of cases still manifest a larger number of neutrophils in the peritoneal dialysis effluent. A preponderance of eosinophils in the peritoneal fluid is seen in a self- limited condition called eosinophilic peritonitis that often follows placement of Tenckhoff catheter and may represent allergy to the tubing. Peritoneal eosinophilia also may be present in fungal and parasitic peritonitis, may be related to chemical and drug (i.e., vancomycin) effects and may be associated with icodextrin.Gram staining of the fluid reveals organisms in the 9% to 50% of cases[3].Peripheral leukocytosis is a poor indicator of for peritonitis in this group of patients. Blood cultures are rarely positive, in contrast to 30% to 50% positive rate in other types of intra-abdominal infections [24].Tranaeus and colleagues in a study of 128 CAPD patients, the initial white cells count (WCC) of the dialysate was less than 100x10(6)/L in 10% of the episodes and showed a predominance of mononuclear cells in 15%, and turbidity used as the sole criterion for the diagnosis of peritonitis [25].

Peritonitis with negative cultures occurs in 5% to 10% of cases. Constant flow of dialysis fluid into and out of the peritoneal cavity dilutes the microbial density and may lower falsely the rate of positive results of the dialysate culture.Negative cultures may also result from infection with fastidious organisms, from previous antimicrobial treatment, or from inadequate culture techniques (e.g. the collection of too little effluent).Culturing the sediment after centrifuging 50ml of effluent dialysate or placing 5 to 10 ml of each of the two blood culture bottles will enhance the recovery rate of the organisms [4]. All cultures to be performed aerobically. Fungal, mycobacterial and anaerobic cultures to be performed if clinically indicated (e.g., negative aerobic cultures).Causes of turbid dialysate, such as hemorrhage, fibrin or other proteins, chylous ascites, and prolonged dwell time should be considered if the leukocyte count is below 300 to 500 cells/mm^3[24].

Tranaeus and colleagues contended that the proportion of negative dialysate cultures was 2% after the introduction of pre-culture membrane filtration. Tunnel infection as a cause of peritonitis was more frequent in episodes due to *Staphylococcus aureus* than in episodes due to coagulase negative *staphylococci*(CNS)(p=0.009).Peritonitis caused by CNS were followed by a milder course than other organisms(p=0.02)[25].

Bibashi and colleagues in a study of 422 CAPD patients evaluated for fungal peritonitis. The diagnosis of peritonitis was based on clinical manifestations (abdominal pain, nausea, and fever) and a cloudy appearance of the dialysate effluent (DE), with WBC count of>100 cells/mm^3,(with neutrophil predominance). Diagnosis was confirmed by isolation of fungi from >1 DE sample [26].In the diagnostic approach to ESP, four aspects need to be evaluated:[27].(1) clinical diagnosis(2) radiological diagnosis(3) pathological diagnosis, and(4) predictive tests.

Radiologic imaging studies are neither specific nor particularly helpful in the diagnosis of peritoneal dialysis- associated peritonitis. Small amounts of free intraperitoneal air, at times, discovered in asymptomatic cases
[24].

V. Treatment

There is a lack of comparative, prospective clinical trials; no antimicrobial regimen is superior to another. After cultures are obtained, initial antimicrobial therapy should be based on the results of Gram staining, or, if Gram stain is not helpful, directed against the most likely pathogens. A reasonable initial empirical regimen would be vancomycin in combination with aminoglycoside. Vancomycin is preferable to a

cephalosporin because of frequency of β-lactam resistance (i.e., methicillin resistance in staphylococci,with a predictive resistance to cephalosporins as well.Alternatively,ceftazidime,cefepime,acarbapenem, or a fluoroquinolone can be used in place of an aminoglycoside for empirical coverage of gram negative organisms. Initial antibiotic choice should be modified, if necessary, after culture results are obtained. Because*P.aeruginosa* peritonitis is associated with high rates treatment failure and relapses, it may be treated withcombination of agents active against the infecting strain, in addition to catheter removal [24].First generation cephalosporin should not be used as first line antibiotic in the treatment of CAPD peritonitis [25].

The minimal therapy needed for dialysis-related peritonitis has not been determined, but usual duration ranges from 10 days to 3 weeks. Most patients with CAPD-associated peritonitis exhibit clinical improvement within 48 to 96 hours after initiation of antimicrobial therapy. If signs and symptoms of peritonitis persists after 96 hours of therapy, reevaluation is warranted; the possibilities of resistant pathogens, unusual organisms (e.g., mycobacterial, fungal), and others intra-abdominal processes should be considered [24].

Fungal peritonitis usually caused by *Candida albicans*,has been treated with amphotericin B,although fluconazole and the echinocandins may be reasonable alternatives[28].Some molds, including*Fusarium*spp, may be resistant to amphotericin B, however, if CAPD is continued, amphotericin B should be given intraperitoneally, but it can cause appreciable abdominal pain when given by this route. Most patients with CAPD-associated fungal infection fail to respond unless catheter is removed; amphotericin B should be given intravenously for 10 days after catheter removal. Oral and intravenous fluconazole penetrates adequately into the peritoneal fluid to treat peritonitis in CAPD recipients after catheter has been removed. The use of echicandins antifungal agents is less documented. Flucytosine is not recommended in azotemic(uremic) patients because of potentially lethal toxicity to the colon and bone marrow. Ketoconazole is rarely indicated.

Additional non-antimicrobial interventions such as routine peritoneal lavage, the use of fibrinolytic agents, and the installation of intraperitoneal immunoglobulins have not been proved beneficialand, therefore serve no role in the management of peritoneal-dialysis-associated peritonitis [29].

Catheter removal is necessary in 10% to 20% of patients. The indications for catheter removal include persistent infection at the skin exit site or tunnel; fungal, fecal or mycobacterial peritonitis; *P.aruginosa* peritonitis; persistent peritonitis; recurrent peritonitis with the same organism; and catheter malfunction (e.g., poor flow).The catheter also should be removed in patients with intraperitoneal abscess. Use of oral or intraperitoneal antibiotics has not been shown to be effective in preventing peritonitis during peritoneal dialysis.

An antibiotic given just before placement of the peritoneal catheter may decrease the incidence of peritonitis and wound infection. Antibiotic prophylaxis has been suggested for patients before extensive dental procedures(although peritonitis caused by dental flora is unusual) and before colonoscopy with polypectomy[4].In addition the topical mupirocin has been used to eliminate nasal carriage with *S.aureus* but has not yet been shown to reduce significantly the incidence of CAPD related peritonitis[30].Advances in CAPD instrumentation, such as titanium adapters, connectors systems with disinfectant, and in-line filters, may decrease the frequency of peritonitis but add to overall cost of CAPD[24].

VI. Prognosis

The prognosis of peritonitis in dialysis recipients is generally favorable. Death is generally not thought of as a significant risk for mortality; however in one retrospective study, death occurred in 6% of 565 patients with 693 episodes of peritonitis [31].The duration of illness and positive peritoneal fluid cultures after institution of antimicrobial therapy is usually 1 to 4 days. Some infections, especially infections caused by *S.aureus,Pseudomonas*spp.,or fungus resolve more slowly, however, and may cause relapse more frequently.

Adequate levels of antimicrobials agents necessary to treat peritonitis successfully can be obtained in the peritoneal fluid by either the systemic or the peritoneal route. Because CAPD-associated peritonitis is a localized infection, however, the intraperitoneal route is preferred and in fact has been found to be superior to the intravenous route [29].The increased use of intraperitoneal antibiotic therapy for peritonitis has allowed most patients to be treated on an ambulatory basis. Hospitalization is indicated for patients who are severely ill or who are unable to manage the administration of intraperitoneal antibiotic at home. Although a variety of dosages and drugs can be found in the literature, the initial dosages recommended, by Priano and colleagues contend that intraperitoneal administration results in effective peritoneal fluid drug concentration [4].Subsequent dosing is used to maintain these levels. The aim of the dosing regimen is to maintain a concentration of the drug in the peritoneal cavity fluid greater than the MIC of the offending pathogens for most if not all, of the dosing interval. However, intermittent dosing regimens (antimicrobials given once daily) and continuous dosing regimens (given in each exchange) have been found to produce largely equivalent results [29].

With intermittent dosing the antimicrobial must dwell for at least 6 hours. Physicians must exercise caution when reviewing the MIC and minimal bactericidal concentration (MBC) data, because these concentrations have been markedly increased when peritoneal dialysis effluent is used as the in vitro growth medium [30].

Symptoms of acute peritonealdialysis.Incidence of peritonitis during acute peritoneal dialysis has remained stable since the 1980s.Innovation in technique, which began during the 1980s, reduced the rate of peritonitis from 50% to lower levels. These innovations include closed-drainage systems, small-bore catheters, limitation of dialysis not more than 72 hours. Incorporation of Millipore filters into the tubing, and development of closed automatic systems. Also the use of dry-heat incubators to warm the dialysate decreases the risk of contamination that may occur when water baths are used for this purpose [24].Some authorities have recommended that cultures of dialysate be obtained every 8 to 24 hours during acute peritoneal dialysis and its termination. Culture of dialysate from the last exchange is more useful than culture of the catheter tip at end of dialysis because the catheter tip is frequently contaminated at the time of its removal. Results ofthese routinecultures, in the absence of symptoms or cloudy fluid, are of doubtful value for initiation of therapy [24].

VII. Conclusion

CAPD is the main form of therapy for end-stage renal disease, and peritonitis remains the major complication of CAPD. Dialysate fluid turbidity can be used as the criterion for the initial diagnosis of peritonitis. First generation cephalosporin is not a good choice as first line antibiotic in the therapy of CAPD peritonitis.

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