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Research Article

Peritonitis related to Peritoneal Dialysis-Analysis of Microbiological Pathogens

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Abstract

The purpose of this study was to summarize the pathogens that cause peritoneal dialysis (PD)-associated peritonitis and to identify risk factors for PD-associated peritonitis. This retrospective study included 84 end-stage renal disease (ESRD) patients receiving PD therapy. Patients were categorized into two groups: the peritonitis group and non-peritonitis group. Clinical data and laboratory tests were collected from medical records. The multivariate logistic regression model was used to evaluate associations between PD-associated peritonitis and potential risk factors. PD-associated peritonitis occurred 54 times in 41 patients. The most frequently identified pathogen was Gram-positive cocci (57.78%). Multivariate logistic regression analysis showed that serum albumin, blood phosphorus, and gastrointestinal disorders were significantly correlated with PD-associated peritonitis. Staphylococcus spp. and E. coli infections were significantly associated with refractory peritonitis. Anemia, nutritional status, biological status of blood phosphorus concentrations, and nutrition status (albumin) were risk factors that influenced the development of peritonitis in our patients with peritoneal dialysis. Secondary hyperparathyroidism and the length of peritoneal dialysis treatment or the adequacy of dialysis had no statistical significance.

Keywords: Peritoneal dialysis; peritonitis; pathogens

Abbreviations: PD: Peritoneal Dialysis; ESRD: End-Stage Renal Disease; HD: Hemodialysis; CVI: Cerebrovascular Incident; CVD: Cardiovascular Disease; Hgb: Hemoglobin; PTH: Parat: Hormone; P: Phosphorous; Ca: Calcium

Introduction

Peritoneal dialysis (PD), hemodialysis (HD), and kidney transplantation are the renal replacement therapy options for patients with end-stage renal disease (ESRD). PD is associated with many complications including catheter tip migration, hypoproteinemia, peritonitis, pleural effusions, and water and electrolyte disturbance. PD-related infections, such as peritonitis, exit-site infection, and tunnel infection, are serious complications for patients with PD [1]. These complications may lead to technique failure, ultrafiltration failure, and inadequacy which forced PD patients to transfer to HD, significant morbidity, and even mortality [2]. The source of PD-related infections included periluminal or intraluminal contamination, bacterial translocation from the bowel or vagina, and hematogenous dissemination. Factors associated with PD-related peritonitis included old age, smoking, obesity, diabetes, hypoalbuminemia, hypokalemia, absence of vitamin D supplements, poor training, previous exit-site infection, and previous peritonitis episodes. The most common microbial cause of PD-associated peritonitis is a gram-positive pathogen [3].

However, gram-negative pathogens, especially Pseudomonas aeruginosa and fungi, are associated with prolonged infection, worse outcomes, and more PD failure. Acute peritoneal dialysis is associated with a high incidence of peritonitis (0.5–4%), and in

the late 1970s, the incidence of peritonitis in patients with chronic peritoneal dialysis was six episodes per year [4]. Sterile peritonitis is non-infectious peritonitis due to the leakage of sterile body fluids into the peritoneum (blood, gastric acid, bile, urine, pancreatic secretion). The symptomatology of peritonitis is linked to the causal trigger, as an entity of inflammation and/or diseases. The knowledge of the pathogenesis of infections associated with PD, possible sources, and reservoirs of potential causes are the basis for defining effective protocols, i.e., guidelines for the prevention and control of infections associated with PD [5]. Infection of the catheter exit site and "tunnel" infection are the basic types of infections [6]. Despite technological innovations (automatic PD) in the field of cysts and solutions for PD, better patient education, and the introduction of preventive measures, peritonitis remains the leading complication of PD.

It is manifested by: diffuse sensitivity of the abdominal wall (70%), blurring of dialysis fluid with leukocytes > 100/mm3 (granulocytes > 50%), and isolation of the dialysis fluid causative agent. For the initial diagnosis of peritonitis, two of the three listed criteria must be satisfied by guidelines [7]. The microbiological diagnosis of peritonitis implies: that the dialysate culture should be taken before susceptible peritonitis, and the first blurred bag is the best sample (50 ml of dialysis); delaying a few hours from the sampling time to the time of planting; staining of Gram-negative sediment from the dialysis bag proves the presence of microorganisms in 20–30% of cases; microbiological cultivation of a dialysis sample for determining the cause, and antibiotic therapy. Laboratory signs of peritonitis in patients on peritoneal dialysis are> 100 Le/mm3 and neutrophil dominance (> 50%); lymphocyte domination in fungal peritonitis; tunnel infection (10%) and less than < 100 Le/mm3); leukocytosis 10000-15000 Le [8]. "Tunnel" infection of the exit site may be affected by erythema, edema, and skin sensitivity above the pathway of the catheter. Many authors have evaluated the role of various catheter implantation techniques and catheter types in lowering the risk of peritonitis in patients [9].

Indications for catheter removal are refractory peritonitis; relapse peritonitis; peritonitis associated with infection of catheter exit site, i.e., "tunnel" infection; fungal peritonitis; repeated peritonitis caused by mycobacteria or multiple enteric microorganisms. After the adequate diagnosis of peritonitis (recommended criteria for diagnoses), it is decided to treat it with appropriate antibiotics: first empirical therapy, and later it is adjusted to antibiogram. The duration of therapy, if the effluent is rapidly clear, is about two weeks. In cases where the response to therapy is not adequate, the removal of the peritoneal catheter is advised five days after the treatment begins [10,11]. The aim was to analyze the incidence of peritonitis and to determine the differences between patients with and without peritonitis and catheter infection. In this study, we retrospectively reviewed the clinical data of ESRD patients who received PD therapy in our hospital and summarized the pathogens responsible for PD-associated peritonitis in these patients. Also, the purpose of this study was to identify risk factors for PD-associated peritonitis, and the results provide valuable clinical information for preventing PD-associated peritonitis and improving survival among PD patients.

Methods

Patients

Retrospectively, 84 patients were treated with continuous ambulatory peritoneal dialysis 2015–2023 at the Clinic for Nephrology at the Clinical Center University of Sarajevo, Bosnia and Herzegovina. The study was performed following the Declaration of Helsinki, with the approval of the local Ethics Committee on Human Research, and informed consent was obtained from each study participant. The diagnosis of peritonitis was made by the recommended guidelines from the above references. All patients started treatment with empirical therapy according to the guidelines for the treatment of peritonitis in patients on PD, or if it was relapsed to earlier sensitivity, and upon the arrival of the dialysate culture, the antibiotic was changed to the antibiogram. Peritonitis was treated for two to three weeks depending on the cause and rate of withdrawal symptoms (one peritonitis was treated for more than three weeks with the protection of fungi, and two episodes caused by the Candida were recorded).

Clinical and laboratory parameters

The diagnosis of peritonitis was based on the clinical picture e.g., turbid dialysis fluid, abdominal pain, sensitivity of the abdomen to palpation, high body temperature, vomiting, fever, and diarrhea. The number of leukocytes in the sediment of dialysate, the findings of PD culture, and the signs of inflammation such as C-reactive protein, the number of leukocytes, etc. Preliminary results were known after two to three days, and definitive after five days of sewing. First-generation cephalosporins as prophylactic antibiotics were administrated before operation in all the patients. Patient training was started soon after Tenckhoff catheter implantation, followed by annual training and retraining after each peritonitis episode. No routine screening was conducted for nasal Staphylococcus aureus colonization. Catheter wound care was performed by the patient or their caregiver using a clean dressing with aqueous povidone-iodine and normal saline. There was no routine wound care with topical mupirocin, gentamicin cream, or ointment.

The patients will have monthly follow-ups of serum biochemistry data such as albumin, sodium, and potassium. Iron profiles, uric acid, serum calcium, and phosphate were followed every 3 months. The peritoneal dialysate was collected and analyzed when the patient reported symptoms associated with peritonitis, such as cloudy dialysate, abdominal pain, or fever. Dialysate was cultured, if there was elevated leukocyte count (>100 cells/mL) or polymorphonuclear neutrophil predominance in the dialysate effluent. An empirical intraperitoneal antibiotic was administrated soon after culture collection. The choice of antimicrobial agent was at the discretion of attending clinicians. Hospitalization was suggested for those with high fever and toxic signs, severe abdominal pain, and fluid overload. Dialysate cultures and fluid analysis were repeated if there was no clinical improvement after antibiotic treatment for 5 days.

Adequate chronic PD implies a prescribed dialysis procedure to ensure a good quality of life for the patient, the absence of physical problems, and morbidity and mortality, which are like

those of the healthy population. The most used parameter for the minimum acceptable weekly values of Kt/V that indicates creatinine clearance according to the American National Kidney Foundation Dialysis Outcome Quality Initiatives recommendations in patients on continuous ambulatory peritoneal dialysis are 1.7 L or 60 L/1.73 m2. The statistical methods included the mean values of numerical variables between two populations using the student t-test and Mann–Whitney test: the categorical variables using $\chi 2$ test for contingency tables and the Fisher test, too. This manuscript presents the measures of descriptive statistics: arithmetic mean, standard deviation, frequency, and percentages.

Results

In the observation period, peritonitis was diagnosed in 41 (47.6%) patients, while 17 (21.4%) patients did not have peritonitis and 26 (31%) had "sterile" peritonitis in rest. Table 1 summarizes the clinicopathological features of patients in the peritonitis and non-peritonitis groups. There were significant differences in the presence of diarrhea or constipation between the non-peritonitis and peritonitis groups ($P < 0.05$). There were no significant differences in patient age, gender, etiology, smoking status, PD status, history of coronary heart disease, and history of cerebrovascular disease and incident. Table 2 summarizes the pathogen culture results for PD effluents from patients with PD-associated peritonitis. Of 54 pathogen cultures, positive results were found in 45 cultures (83.33%), including 26 (57.78%) cultures with Gram-positive cocci, 12 (26.67%) cultures with Gram-negative bacilli, and 1 culture with fungi.

Table 1: The clinicopathological features of patients in the non-peritonitis and peritonitis groups.

Risk factors	Non-peritonitis (n = 74)	Peritonitis (n = 41)	P value
Age (y) median (range)	61.0 (17, 81)	56.5 (21, 83)	0.282
Male	34	17	0.105
Female	40	24	
Etiology known	23	11	0.225
Etiology unknown	51	30	
Smoking (yes)	12	4	0.281
PD after HD or kidney transplantation	10	7	0.758
Direct PD	64	34	
Hystori CVD (yes)	10	8	0.610
History CVI (yes)	10	3	0.374
Diarrhea or constipation (yes)	7	11	0.036

Table 2: The pathogen culture results for PD effluents in patients with PD-associated peritonitis.

Gram-negative bacteria	**12**	**22.22**
Escherichia coli	6	11.11
Enterobacter cloacae	1	1.85
Serratiamarcescens	1	1.85
Acinetobacterbaumannii	1	1.85
Enterobacteragglomerans	1	1.85
Klebsiella pneumonia	2	3.70
Gram-positive bacteria	**26**	**48.15**
Staphylococcus epidermidis	7	12.96
Enterococcus faecalis	3	5.56
Staphylococcus aureus	5	9.26
Staphylococcus in chickens	1	1.85
Streptococcus bovis	1	1.85
Staphylococcus haemolyticus	1	1.85
Coagulase-negative staphylococcus	1	1.85
Streptococcus salivarius	1	1.85
Fungi		
Candida parapsilosi	2	3.70
Candida Albicans	1	1.85

Of the three cases with fungal infection, primary fungal infection occurred in only one case, and infection secondary to bacteria occurred in the other two cases. The most frequently identified Gram-positive coccus was Staphylococcus epidermidis (7 cases), and the most frequently identified Gram-negative bacillus was Escherichia coli (6 cases). Eight infections of the outlet were identified during the analyzed period, four of them were associated with peritonitis. The most common causes of infection were Staphylococcus aureus (four patients), Staphylococcus coagulase negative (two patients), Pseudomonas aeruginosa (one patient), and Enterobacter (one patient) (Table 3). Table 4 summarizes the laboratory results for patients with the non-peritonitis and peritonitis groups. There were significant differences in the serum concentrations of albumin, hemoglobin, blood phosphorus, and PTH between the non-peritonitis and peritonitis groups ($P < 0.05$).

Table 3: Causes of infections catheter exit site of peritoneal catheter in patients.

Causative agents of Catheter exit site Infection	Catheter outlet infection	%
Staphylococcus aureus	4	44.5
Staphylococcus spp.	2	22.2
Pseudomonas	1	11.1
Enterococcus	1	11.1
Total	8	100

Table 4: The laboratory results for patients in the non-peritonitis and peritonitis groups.

Factors	Peritonitis	Non-peritonitis	P
Albumin (g/L)	26.70 (18.0, 37.9)	35.50 (20.7, 50.7)	<0.001
Ca (mmol/L)	2.10 (1.30, 2.78)	2.00 (1.22, 2.96)	0.119
P (mmol/L)	1.32 (0.66, 2.18)	1.70 (0.66, 4.69)	<0.001
PTH (mmol/L)	81.38 (2.90, 585.40)	240.60 (12.90, 1501.00)	<0.001
Erythrocyte	3.09 ± 0.68	3.67 ± 0.97	0.013
Hgb (g/L)	92.00 (28, 125)	83.50 (46, 141)	0.033
Iron	10.04 ± 4.13	12.5 ± 4.04	0.004

Data is presented as mean (range).

There were no significant differences in the serum creatinine and blood calcium concentrations between the two groups ($P > 0.05$). We further used the multivariate logistic regression model, including risk factors such as gastrointestinal disorders, serum albumin, hemoglobin, erythrocyte, blood iron concentration, blood phosphorus concentration, and PTH, for predicting PD-associated peritonitis. Based on the multivariate logistic regression analysis, erythrocyte, hemoglobin, iron and PTH were not significantly associated with PD-associated peritonitis ($P > 0.05$). Serum albumin ($\beta = -0.208$, $P < 0.01$), blood phosphorus concentration ($\beta = -1.732$, $P = 0.001$), and gastrointestinal disorders ($\beta = 1.624$, $P = 0.043$), were significantly associated with PD-associated peritonitis (Table 5).

Table 5: Multivariate logistic regression analysis of the risk factors for PD-associated peritonitis.

Factors	Regression coefficient β	SE	P	OR	95%CI
Albumin	−0.208	0.047	<0.001	0.812	0.741-0.891
Gastrointestinal disorders	1.624	0.801	0.043	5.075	1.056-24.398
Phosphorus	−1.732	0.528	0.001	0.177	0.063-0.498

Discussion

During the analysis period, the most common causes of peritonitis in our patients were: Staphylococcus aureus, Staphylococcus coagulase-negative, and Escherichia coli. The incidence of peritonitis decreased in one for eight and 24 months of the treatment. The significance of peritonitis prevention, quality patient training for an independent examination of treatment techniques, and technological innovations in the field of cysts and solutions further reduce the incidence of peritonitis. The incidence of peritonitis in patients in Canada was one episode at 26 patient months (1996–2005) [12]. Peritonitis is a common PD-associated complication that can lead to hospitalization and PD failure. The main cause of PD-associated peritonitis is due to inappropriate handling. However, many risk factors can increase the susceptibility of PD patients to peritonitis. In this study, we found that serum albumin and blood phosphorus concentrations in addition to diarrhea or constipation were risk factors for PD-associated peritonitis.

The identification and control of risk factors for PD-associated peritonitis are important for the prevention and treatment of PD-associated peritonitis. PD-associated peritonitis can be caused by many pathogens, such as bacteria and fungi. In this study, 9 (16.67%) of the 54 pathogen cultures were negative, and this negative pathogen culture rate is consistent with the recommended target for culture-negative peritonitis episodes is <15% set by the International Society for Peritoneal Dialysis (ISPD) [13]. In addition, we found that the most frequent pathogen was Gram-positive cocci (57.78%), followed by Gram-negative bacilli (26.67%). The most frequently identified Gram-positive coccus was Staphylococcus epidermidis, and the most frequently identified Gram-negative bacillus was Escherichia coli. These results are consistent with those of previous reports in the literature [14]. PD-associated peritonitis caused by Gram-positive coccus is mainly due to inappropriate PD handling.

In this study, all patients who were trained for appropriate use of PD lived in the city and did not share a room with others. However, Gram-positive coccus was still the most common pathogen responsible for PD-associated peritonitis in our patients. Therefore, it is important to thoroughly train every PD operator to improve appropriate PD handling. In addition, enterobacteria-associated peritonitis commonly occurs in patients with intestinal disorders or diabetes. Moreover, diabetes patients often have intestinal disturbance, chronic inflammation, or nutritional deficiency, which can lead to diarrhea or constipation. Therefore, PD-associated peritonitis caused by enterobacteria is commonly observed in patients with diarrhea or constipation. "Sterile" peritonitis or culture-negative peritonitis (25.8%) was more commonly reported in our patients, than in patients of another authors-Szeto et al. [15] (17.9%).

In our patients, there were fewer outbreaks of infection during the analyzed period, compared to the other authors, and associated with severe peritonitis. In Australia, Govindarajulu S et al. [16] found that 14% of peritonitis is caused by Staphylococcus aureus. In our patients, the frequency of peritonitis caused by Staphylococcus aureus was 9.26% because it is the cause of severe peritonitis with a worse prognosis. In Australian patients, Pseudomonas infections were less common (2.1%), with E. coli (6.3%) and Klebsiella (4%) more often than in our patients. Fungal infections were not frequent in our center: only three patients had this infection, while experts in Australia accounted for 3.1% of fungal peritonitis. For PD-associated peritonitis, fungal infection is relatively rare. Fungal peritonitis is severe and usually occurs only after the use of broad-spectrum antibiotics for the treatment of bacterial peritonitis.

Most often the high incidence rate of fungal infection is likely secondary to repeated bacterial infection because cases with fungal peritonitis were secondary to repeated bacterial infection. These findings suggest that PD patients with repeated bacterial peritonitis are susceptible to fungal infection. We thought that the relatively large number of patients with fungal peritonitis may be associated with secondary after antibiotic treatment. A particular problem in all patients on dialysis is anemia [17]. Previous studies showed that patients on PD had anemia, but less pronounced anemia syndrome than patients undergoing repeated hemodialysis. This beneficial effect of peritoneal dialysis can be explained by higher erythropoietin concentrations, reduced concentration of erythropoiesis inhibitors, and higher quality of nutrition (respectively nutritional status). It is now believed that a significant difference in the severity of anemia among patients on treatment with PD and HD was associated with better clearance of middle molecules, which are essential inhibition factors of the same.

During the analyzed period by examining the impact of anemia on the development of peritonitis, we found that anemia was a significant risk factor for the development of peritonitis. The other factors that increase the risk of peritonitis include age, diabetes mellitus, obesity, cardiovascular disease, depression, catheter linkage, and/or catheter infections [18]. Prevention of peritonitis associated with peritoneal dialysis represents a high treatment priority [19]. Clinical practice patterns are very different today. Intravenous vancomycin may reduce the risk of early peritonitis and peri-operative treatments. Antifungal prophylaxis with oral nystatin or oral fluconazole may also reduce the risk of fungal peritonitis. Another antimicrobial therapy has not shown adequate efficacy. In Japan, developing effective outpatient protocols for peritonitis treatment and ready and prompt access to home-administered intra-peritoneal antibiotics may reduce the costs associated with peritonitis treatment and peritoneal dialysis therapy [20].

The authors suggest that the biological status of iron in patients on peritoneal dialysis may be a risk factor for the development of infectious peritonitis (improving the growth of bacteria through transferring iron) [21]. Also, from our results about peritonitis's influence on mortality, Tekkarismaz et al. [22] have shown that peritonitis did not reduce patient survival. Malnutrition is a common complication in PD patients and is closely associated with PD incidence and mortality [23]. The serum albumin concentration has been recommended by the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (K/DOQ) for assessing the nutritional status of patients [24]. In this study, we found that the serum albumin concentration was significantly correlated with PD-associated peritonitis. Furthermore, multivariate logistic regression analysis showed that the serum albumin level was negatively associated with PD-associated peritonitis, suggesting that a low level of serum albumin is associated with a higher risk for PD-associated peritonitis.

Consistent with our findings, Ozturk et al. [25] reported that a decrease in serum albumin is associated with an increased risk for PD-associated peritonitis. Based on our findings, correction of malnutrition, increased protein intake, and improved appetite are important for preventing and treating PD-associated peritonitis. CKD patients commonly have hyperphosphatemia, which can lead to secondary hyperparathyroidism, excessive deposition of calcium and phosphorus, disrupted vitamin D metabolism, and renal osteodystrophy. In this study, we found that blood phosphorus concentrations were significantly correlated with PD-associated peritonitis. Furthermore, logistic regression analysis showed that blood phosphorus concentrations were negatively correlated with PD-associated peritonitis, suggesting that low blood phosphorus

concentrations are associated with a higher risk for PD-associated peritonitis. One explanation for the inverse relationship between serum phosphorous and peritonitis incidence may be the association between malnutrition and low blood phosphorous concentration.

Diarrhea or constipation occurred more frequently in the peritonitis group than in the non-peritonitis group, suggesting that gastrointestinal disorder is a risk factor for PD-associated peritonitis. Furthermore, multivariate logistic regression analysis showed that diarrhea and constipation were positively correlated with PD-associated peritonitis, suggesting that the presence of diarrhea or constipation increases the risk for PD-associated peritonitis. It has been reported that peritonitis can be caused by the transmural migration of intestinal microbes [26]. In ESRD patients with uremia, the intestinal wall exhibits pathological changes that allow microbes to enter the abdominal cavity to induce peritonitis via the lymph nodes or intestinal wall during intestinal infection and diarrhea or constipation. Constipation is very common in ESRD patients but is often ignored. It is important to guide PD patients to defecate normally to prevent constipation and thereby reduce the incidence of peritonitis caused by intestinal microbes.

Conclusion

In PD-related peritonitis, gram-positive cocci were major pathogens. About three-quarters of causative pathogens were susceptible to empirical antimicrobial regimens (cefazolin and gentamicin) recommended by ISPD for PD-related peritonitis. Staphylococcus spp. and E. coli infections were significantly associated with refractory peritonitis. Anemia, nutritional status, biological status of blood phosphorus concentrations, and protein status (albumin) were risk factors that influenced the development of peritonitis in our patients with peritoneal dialysis. Secondary hyperparathyroidism and the length of peritoneal dialysis treatment or the adequacy of dialysis had no statistical significance. Further studies with a large sample size across multiple centers are required to confirm our findings.

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None.

Conflict of Interest

No conflict of interest.

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