Peritonitis before Peritoneal Dialysis Training: Analysis of Causative Organisms, Clinical Outcomes, Risk Factors, and Long-Term Consequences

Terry King-Wing Ma, Kai Ming Chow, Bonnie Ching-Ha Kwan, Wing Fai Pang, Chi Bon Leung, Philip Kam-Tao Li, and Cheuk Chun Szeto

Abstract

Background and objectives Peritonitis before peritoneal dialysis (PD) training (pretraining peritonitis [PTP]) is an uncommon event. The study aim was to examine the causative organisms, clinical outcomes, risk factors, and long-term consequences of PTP.

Design, setting, participants, & measurements In this single–center, retrospective, observational study involving all incident patients on PD who developed PTP between 1998 and 2012, we examined the causative organisms, primary response rate, complete cure rate, risk factors, and associations of PTP with peritoneal equilibration test (PET) and patient survival. For each patient in the PTP group, the patients who underwent catheter insertion immediately before and after the index case were identified as controls.

Results Among 1252 incident patients on PD, 52 (4.2%) patients developed PTP, and 104 patients were identified as controls. The two groups were similar in age, sex distribution, comorbidities, and residual renal function, but the PTP group had significantly lower hemoglobin and serum albumin. Patients were followed up for a median of 37.5 months (interquartile range [IQR], 16.3–62.2 months). The most common causative organisms of PTP were *Staphylococcus aureus* (30.8%) and polymicrobial (21.2%); 25% had negative growth. The primary response and complete cure rates were 82.7% and 78.8%, respectively. In the PTP group, 7.7% of patients died, 9.6% of patients required catheter removal, and PD training was significantly delayed (median =42.0; IQR, 26.0–65.8 days versus 27.5; IQR, 23.0–35.0 days; P=0.01). Multivariate logistic regression analysis showed that serum albumin was the only predictor of PTP (adjusted odds ratio, 0.89 per 1-g/dl increase; 95% confidence interval, 0.82 to 0.97). There were no differences in PET results and dialysis adequacy (measured around 1 month after PD training). The PTP group had significantly worse patient survival (median =41.2; IQR, 21.8–60.5 months versus 55.8; IQR, 40.4–71.2 months; P=0.02). Technique failure occurred in 11.5% and 10.6% of patients in the PTP and control groups, respectively.

Conclusions *S. aureus* is the most common causative organism of PTP. Nutritional interventions in patients who are hypoalbuminemic before catheter insertion deserve additional study.

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Department of Medicine and Therapeutics, Carol and Richard Yu Peritoneal Dialysis Research Centre, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, Hong Kong

Correspondence: Dr. Cheuk Chun Szeto, Department of Medicine and Therapeutics, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, NT, Hong Kong, China. Email: ccszeto@cuhk.edu.hk

Introduction

Peritoneal dialysis (PD) is the first-line RRT for patients with ESRD in Hong Kong (1). Despite well established guidelines for prevention (2) and treatment (3) of PD-related infections, peritonitis remains a major challenge to nephrologists and a significant cause of morbidity and mortality in patients on PD (4). In incident patients on PD early peritonitis is associated with significant negative effects on long–term patient and technique survival (5–7).

Peritonitis before PD training (pretraining peritonitis [PTP]) is an uncommon event. Also, most centers only capture peritonitis after patients have completed PD training and started on maintenance dialysis. Therefore, there is a dearth of literature on this subject. The aim of this study was to examine the causative organisms, clinical outcomes, risk factors, and long-term consequences of PTP.

Materials and Methods

Patient Selection and Data Collection

This was a single–center, retrospective, observational study involving patients with ESRD who had PD as their first modality of RRT in Prince of Wales Hospital from January 1, 1998 to December 31, 2012. Patients with history of chronic hemodialysis (HD) and kidney transplantation were excluded. PTP was defined as peritonitis after catheter insertion and before PD training. For each patient in the PTP group, the patients who underwent catheter insertion immediately before and after the index case were identified

as controls. Clinical information was obtained from computerized records in the Hospital Authority Clinical Management System and Prince of Wales Hospital Renal Registry followed by manual review of medical and nursing records. Baseline demographic and clinical characteristics at the time of catheter insertion, including age, sex, cause of ESRD, body mass index (BMI), hepatitis status, comorbidities, Charlson comorbidity score, hemoglobin, serum albumin, and residual GFR, were recorded. Normalized protein nitrogen appearance and lean body mass (LBM) were measured during PD training. eGFR was calculated as the average of 24-hour urinary urea and creatinine clearance (8). Normalized protein nitrogen appearance was determined by the formula by Bergström *et al.* (9). LBM or fat-free, edema–free body mass was measured by creatinine kinetics according to the formula of Forbes and Bruining (10) and presented as percentage of the ideal body weight.

Catheter Insertion and Postoperative Care

After informed consent, catheters were inserted by fully trained nephrologists in a dedicated ward–based procedure room using the open dissection surgical technique under local anesthesia as previously described (11). All catheters were double–cuffed coiled Tenckhoff catheters. All patients received intravenous cefazolin before operation as prophylactic antibiotic. Intravenous vancomycin was used in patients with penicillin or cephalosporin allergy. Povidone iodine was used for skin preparation intraoperatively, and povidone iodine ointment was applied to the exit site at the end of surgery. The wound and exit site were covered with absorbent dressings and kept intact for at least 1 week. Subsequent wound and catheter care were undertaken by trained PD nurses according to standard protocols. Patients were educated by trained PD nurses to keep the main wound covered before removal of stitches, and the exit site should be covered and not get wet before PD training. Screening for nasal carriage of *Staphylococcus aureus* was not performed routinely, and prophylactic topical antibiotic to exit site was not used. Immediately after surgery, the peritoneal cavity was flushed with 1 L dialysis fluid until clear peritoneal dialysis effluent (PDE) was observed. If continuous dialysis was not required, the catheter was capped off by trained PD nurses. Low–volume intermittent peritoneal dialysis (IPD) using straight set and 1-L dwell was continued in the supine position if dialysis was indicated. In all patients, PD training was arranged 3–4 weeks after catheter insertion. In the early study period, patients were regularly followed up at least once a week after catheter insertion. Volume status and serum biochemistry were closely monitored. Patients were admitted to the hospital for manual IPD over 2 days if dialysis was required before PD training. Manual IPD using 1-L dwell was used within 2 weeks of catheter insertion, and patients were switched to machine-assisted IPD with 2-L dwell after 2 weeks of catheter insertion. In the late study period, IPD was performed in a day ward using cyclers in patients who required dialysis before PD training. The usual regime was 8 hours of IPD two times a week. Dwell volume was limited to 1 L in the first 2 weeks of catheter insertion, and 2-L dwell was used after 2 weeks. Before PD training, all patients were dialyzed using conventional lactate–buffered, glucose–based PD solutions. If dialysis was not required, flushing of catheter was performed using standard protocol once weekly. All IPD procedures and flushing of the catheter immediately after surgery and before PD training were performed by trained PD nurses using standard protocols.

Diagnosis and Treatment of Peritonitis

The diagnosis of peritonitis was on the basis of at least two of three of the following criteria: (*1*) abdominal pain or cloudy PDE, (*2*) PDE leukocyte count $>100/\mu l$ with at least 50% neutrophils, and (*3*) positive Gram stain or culture of PDE (12). Bacterial culture of PDE was performed throughout the period in BacTAlert Bottles (Organon Teknika, Durham, NC) according to recommendations of the International Society of Peritoneal Dialysis (3,12–14). Empirical intraperitoneal antibiotics were initiated according to standard protocols in the unit (15). Effective antibiotic treatment was continued for 2 weeks in general and 3 weeks for peritonitis caused by *S. aureus* or *Pseudomonas* or *Enterococcus* species. Metronidazole was added when anaerobic bacteria were isolated. Refractory peritonitis was defined as the failure of PDE to clear after 5 days of appropriate antibiotics. Primary response was defined as resolution of abdominal pain, clearing of PDE, and PDE leukocyte count $<100/\mu l$ after 10 days of antibiotic therapy. Complete cure was defined as resolution of peritonitis without relapse or recurrence within 4 weeks after completion of antibiotics. In general, patients had catheter removal if PDE failed to clear up after 10 days. Patients were switched to temporary HD after catheter removal, and appropriate antibiotic therapy was continued for another 2 weeks. Catheter reinsertion was attempted in all patients, and if it failed, patients were switched to long-term HD.

Risk Factors Analyses

Our center previously showed that diabetes mellitus and initial serum albumin concentration were independent predictors of peritonitis in incident patients on PD (16). There was also a nonsignificant trend toward increased risk of peritonitis in patients with cerebrovascular disease. Different modifiable and nonmodifiable risk factors for peritonitis have been identified from other observational studies and summarized elsewhere (17). In this study, we tested age, sex, BMI, LBM, diabetes mellitus, cerebral vascular disease, ischemic heart disease (IHD), peripheral vascular disease, Charlson comorbidity score, hepatitis status, hemoglobin, serum albumin, and residual GFR as potential predictors of PTP.

Peritoneal Equilibration Test and Dialysis Adequacy

The association of PTP with peritoneal equilibration test (PET) was evaluated. In the PTP group, PET was performed after resolution of peritonitis. In both groups, PET was performed approximately 1 month after completion of PD training using standard methods (18). Dialysate-to-plasma ratios of creatinine at 4 hours was calculated after correction of glucose interference (19). Mass transfer area coefficient of creatinine normalized for body surface area was calculated by the formula described by Krediet *et al.* (20). Dialysis adequacy, expressed as total weekly Kt/V, was determined by standard models (21).

Long-Term Outcomes

Patient survival was administratively censored on July 1, 2015. Censoring events included transfer to long-term HD, kidney transplant, recovery of renal function, loss to follow-up, or transfer to other dialysis centers. Death within 30 days of switching to HD was considered death on PD (22). Technique failure was defined as permanent cessation of PD and transfer to HD. The first episode of peritonitis after completion of PD training was recorded in the control groups for comparison with the PTP group.

Statistical Analyses

Statistical analysis was performed using SPSS (SPSS Inc., Chicago, IL) for Windows (Microsoft, Redmond, WA) software, version 17.0. All data are expressed as means±SDs unless otherwise specified. Baseline demographic data were compared using chi-squared test, Fisher exact test, *t* test, and Mann–Whitney *U* test as appropriate. Univariate and multivariate logistic regression analyses were performed to identify predictors of PTP. Because the control patients were identified by their time of PD catheter insertion without matching of any demographic or clinical characteristics, conventional logistic regression analysis was performed. Factors with $P<0.10$ in univariate analysis were included in multivariate analysis. A backward elimination procedure using $P>0.05$ was performed to identify independent predictors of PTP. A Kaplan–Meier curve was constructed to compare cumulative patient survival. The difference between groups was assessed by log rank test. Univariate and multivariate Cox regression analyses were performed to see whether PTP was an independent predictor of mortality. In addition to the patient group (PTP versus control), the Cox model was constructed by age, Charlson comorbidity score, serum albumin, total Kt/V, and residual GFR. These parameters were selected for construction of the Cox model because of their importance in determining the survival of patients on PD. A *P* value of <0.05 was considered statistically significant. All probabilities were two tailed.

Results

Among 1252 incident patients on PD, 52 patients (4.2%) developed PTP, and 104 patients were identified in the control group. The baseline demographic and clinical characteristics are shown in Table 1. The PTP group had significantly lower hemoglobin and serum albumin. Patients were followed up for a median of 37.5 months (interquartile range [IQR], 16.3–62.2 months). No patients were lost to follow-up.

All patients required IPD before PD training. The median onset time of PTP was 16 days (IQR, 8–27 days), counting from the day of catheter insertion. Among 52 PTP episodes, 12 occurred within the first week, 10 occurred in the second week, and 30 occurred after 2 weeks; 12 of 52 patients (23.1%) had concomitant exit site infection (ESI). In these 12 patients, 10 had *S. aureus* ESI, one had polymicrobial ESI, and one had coagulase–negative *Staphylococcus* (coagulase-negative staphylococcus) ESI. In the PTP group, all patients were admitted to the hospital for treatment of peritonitis. The

Table 1. Baseline demographic and clinical characteristics of patients

Parameters	Peritonitis	Control	*P* Value
No. of patients	52	104	
Age, yr	58.4±12.7	57.8±13.2	0.79
Sex, men/women	36/16	55/49	0.59
BMI, kg/m^2	25.1±5.8	24.2±4.3	0.34
LBM, %	44.4±16.6	40.3±10.4	0.09
Cause of ESRD			
DM	22 (42.3%)	48 (46.2%)	
HT	2 (3.8%)	5 (4.8%)	
GN	11 (21.2%)	25 (24.0%)	
Others	6 (11.5%)	13 (12.5%)	
Unknown	11 (21.2%)	13 (12.5%)	
Comorbidities			
DM	27 (51.9%)	56 (53.8%)	0.87
CVA	17 (32.7%)	25 (24.0%)	0.26
IHD	10 (19.2%)	29 (27.9%)	0.33
PVD	4 (7.7%)	5 (4.8%)	0.48
Hepatitis B	4 (7.7%)	12 (11.5%)	0.58
Hepatitis C	2 (3.8%)	1 (0.96%)	0.26
Charlson score[a]	6.0 (4.0–8.0)	6.0 (4.3–8.0)	0.95
Biochemistry			
Hb, g/dl	8.6±1.7	9.3±1.6	0.03
Albumin, g/dl	3.0±0.6	3.2±0.5	0.03
eGFR, ml/min per 1.73 m^2	3.16±2.82	3.82±2.44	0.21
NPNA, g/kg per d	1.13±0.30	1.16±0.26	0.65

BMI, body mass index; LBM, lean body mass; DM, diabetes mellitus; HT, hypertension; CVA, cerebrovascular disease; IHD, ischemic heart disease; PVD, peripheral vascular disease; Hb, hemoglobin; NPNA; normalized protein nitrogen appearance.
[a]Median (interquartile range).

Table 2. Causative organisms of pretraining peritonitis compared with first peritonitis in the control group

Organisms	Pretraining Peritonitis			Control Overall, *n*=62
	Overall, *n*=52	Within first 2 wk, *n*=22	After 2 wk, *n*=30	
S. aureus	16 (30.8%)	6 (27.3%)	10 (33.3%)	6 (9.7%)
Negative growth	13 (25.0%)	6 (27.3%)	7 (23.3%)	7 (11.3%)
Mixed growth	11 (21.2%)	6 (27.3%)	5 (16.7%)	6 (9.7%)
Gram-negative bacilli	5 (9.6%)	1 (4.5%)	4 (13.3%)	15 (24.2%)
Coagulase-negative *Staphylococcus*	2 (3.8%)	1 (4.5%)	1 (3.3%)	10 (16.1%)
Anaerobes	2 (3.8%)	1 (4.5%)	1 (3.3%)	0
Streptococcus	1 (1.9%)	0	1 (3.3%)	10 (16.1%)
Corynebacterium	0	0	0	4 (6.5%)
Enterococcus	0	0	0	1 (1.6%)
Fungus	1 (1.9%)	0	1 (3.3%)	0
Tuberculosis	1 (1.9%)	1 (4.5%)	0	3 (4.8%)

causative organisms of PTP compared with the first peritonitis in the control group are shown in Table 2. The most common organisms causing PTP were *S. aureus* (30.8%) and polymicrobial (21.2%). A similar pattern was observed for PTP occurring within and after 2 weeks of catheter insertion. Twenty-five percent of patients had culture-negative peritonitis. In contrary, the most common organisms causing the first episode of peritonitis in the control group were Gram-negative bacilli (24.2%), coagulase-negative staphylococcus (16.1%), and *Streptococcus* species (16.1%). In the control group, 15.4% of patients developed early peritonitis within 6 months. The most common organisms were Gram-negative bacilli (18.8%), *S. aureus* (12.5%), and *Streptococcus* species (12.5%).

The primary response and complete cure rates of PTP were 82.7% and 78.8%, respectively. Among nine patients who failed to have primary response, four patients died, and five required catheter removal. Mortality rate of PTP was 7.7%. The causative organisms in patients with fatal PTP were polymicrobial (*n*=3) and fungus (*n*=1). Causes of death were peritonitis, infection other than peritonitis, liver failure, and sudden cardiac death. In five patients who had catheter removal, the causative organisms were *S. aureus*, *Streptococcus* species, anaerobes, polymicrobial, and tuberculosis. The detailed clinical outcomes are shown in Figure 1. As a result of peritonitis, PD training was significantly delayed in the PTP group (median =42.0 days; IQR, 26.0–65.8 days versus 27.5 days; IQR, 23.0–35.0 days; *P*=0.01).

Univariate logistic regression analysis showed that BMI (unadjusted odds ratios [OR], 1.16; 95% confidence interval [95% CI], 1.03 to 1.30; *P*=0.02) and serum albumin (unadjusted OR, 0.89 per 1-g/dl increase; 95% CI, 0.81 to 0.98; *P*=0.02) were predictors of PTP. Multivariate analysis showed that serum albumin was the only significant predictor of PTP (adjusted OR, 0.89 per 1-g/dl increase; 95% CI, 0.82 to 0.97; *P*<0.01) as shown in Table 3. In other words, every 1 g/dl higher serum albumin concentration was associated with 11% lower odds of PTP.

There were no significant differences between the two groups in terms of drainage volume during PET (2.28±0.20 L in the PTP groups versus 2.35±0.19 L in the control group; *P*=0.55), dialysate-to-plasma ratios of creatinine at 4 hours (0.68±0.11 versus 0.64±0.11; *P*=0.83), mass transfer area coefficient of creatinine (10.35±3.96 versus 9.54±4.65; *P*=0.89), dialysate protein concentration (1.10±0.48 g/L versus 0.94±0.37 g/L; *P*=0.06), and Kt/V (1.95±0.57 versus 2.17±0.55; *P*=0.28).

At the end of the study, 38 of 52 (73.1%) patients died in the PTP group, whereas 65 of 104 (62.5%) patients died in the control group. Patient survival was significantly worse in the PTP group (median =41.2 months; IQR, 21.8–60.5 months versus 55.8 months; IQR, 40.4–71.2 months; *P*=0.02 by log rank test) as shown in Figure 2. Univariate and multivariate Cox regression analyses, however, showed that PTP *per se* was not an independent predictor of mortality (multivariate hazard ratio, 1.10; 95% CI, 0.63 to 1.94; *P*=0.70) as shown in Table 4. The most common causes of death in the PTP group were infection other than peritonitis (36.8%), termination of dialysis (26.3%), and IHD/myocardial infarction (15.8%). On the contrary, peritonitis (24.6%) was the most common cause of death in the control group followed by IHD/myocardial infarction (18.5%) and stroke (15.4%). In the PTP group, six patients had

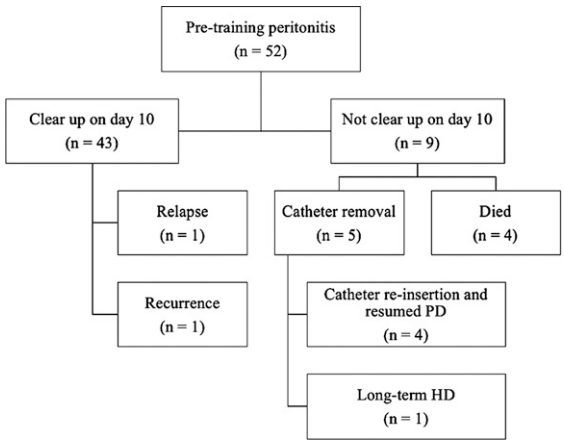

Figure 1. | Clinical outcomes of pretraining peritonitis. HD, hemodialysis; PD, peritoneal dialysis.

Table 3. Univariate and multivariate logistic regression analyses of predictors of pretraining peritonitis

Variable	Univariate		Multivariate	
	Odds Ratio (95% CI)	*P* Value	Odds Ratio (95% CI)	*P* Value
Age, yr	1.01 (0.94 to 1.08)	0.88		
Women	2.32 (0.84 to 6.46)	0.11		
Hepatitis B	1.82 (0.26 to 12.91)	0.55		
DM	1.88 (0.35 to 10.20)	0.46		
IHD	2.72 (0.53 to 13.89)	0.23		
CVA	1.37 (0.24 to 7.80)	0.72		
PVD	1.04 (0.16 to 6.73)	0.97		
Charlson score	1.29 (0.67 to 2.51)	0.45		
BMI, kg/m^2	1.16 (1.03 to 1.30)	0.02	1.07 (0.99 to 1.17)	0.10
Hb, g/dl	0.86 (0.65 to 1.14)	0.29		
Albumin, g/dl	0.89 (0.81 to 0.98)	0.02	0.89 (0.82 to 0.97)	<0.01
eGFR, ml/min per 1.73 m^2	1.02 (0.80 to 1.30)	0.90		
LBM, %	1.05 (0.99 to 1.10)	0.11		

95% CI, 95% confidence interval; DM, diabetes mellitus; IHD, ischemic heart disease; CVA, cerebrovascular disease; PVD, peripheral vascular disease BMI, body mass index; Hb, hemoglobin; LBM, lean body mass.

technique failure, two were transferred to other centers, one had kidney transplantation, and one had recovery of renal function. In the control group, 11 had technique failure, 15 had kidney transplantation, two had recovery of renal function, and one was transferred to another center. Technique failure rates were 11.5% and 10.6% in the PTP and control group, respectively.

Discussion

To our best knowledge, this is the first study to examine patients suffering from PTP. We found that *S. aureus* and polymicrobial peritonitis accounted for >50% of all patients. This was different from the control group as a whole as well as control patients who developed early peritonitis within the first 6 months of PD. In the PTP group, all *S. aureus* was methicillin sensitive. Previous studies showed that nasal, skin, and pericatheter colonizations of *S. aureus* were important risk factors for developing *S. aureus* peritonitis, and prophylaxis using oral rifampin or mupirocin ointment in the nares or exit site significantly reduced the rate of exit site infection and possibly, peritonitis (23). Screening of *S. aureus* carriage was

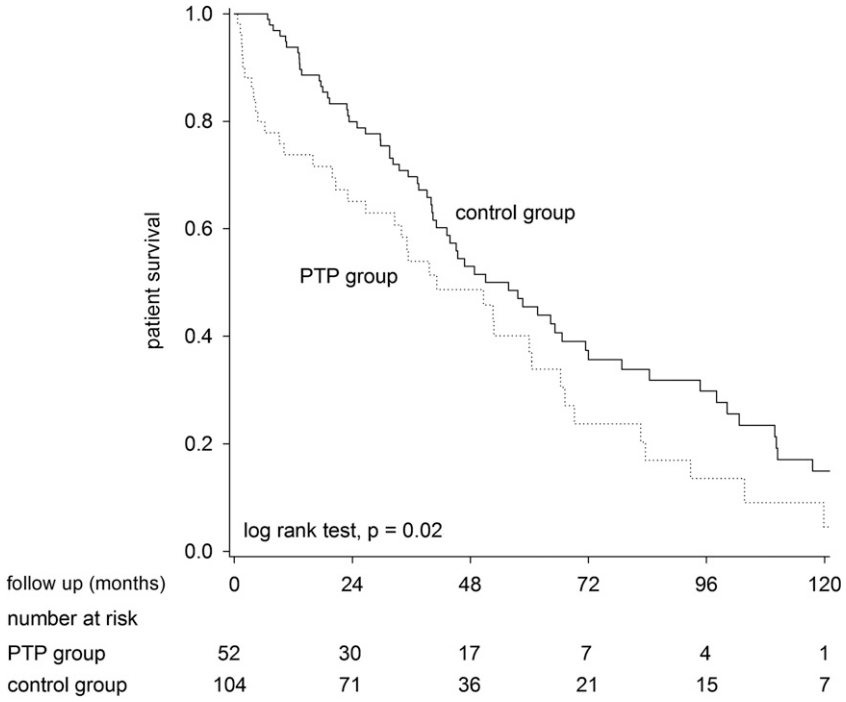

Figure 2. | Kaplan—Meier curve of patient survival. PTP, pretraining peritonitis.

Table 4. Multivariate Cox regression analysis of predictors of mortality

Variable	AHR	95% CI	*P* Value
Charlson score	1.17	1.08 to 1.27	<0.001
Serum albumin (for every 1-g/dl increase)	0.95	0.91 to 0.99	0.02
Pretraining peritonitis[a]	1.10	0.63 to 1.94	0.70

AHR, adjusted hazard ratio; 95% CI, 95% confidence interval.
[a]Compared with the control group.

not routinely performed in our center during the early study period. However, we reported that, in patients suffering from *S. aureus* peritonitis, those with a history of recent hospitalization had a higher risk for isolation of methicillin–resistant *S. aureus* (24). Screening for methicillin–resistant *S. aureus* in high-risk patients was performed in later years of the study. As a result, we were unable evaluate the effect of *S. aureus* carriage on PTP because of unavailability of data. Among 16 patients with PTP and *S. aureus* peritonitis, four had concomitant *S. aureus* ESI, and one had polymicrobial ESI. In these 16 patients, the catheters were inserted by eight different nephrologists. In four patients with *S. aureus* ESI and PTP, the catheters were inserted by four different nephrologists. Although nasal carriage of *S. aureus* by an operator might be a potential source of infection, we found no evidence that these peritonitis episodes could be attributed to any particular operator.

One fourth of patients in the PTP group had culture-negative peritonitis. This was substantially higher than our previously reported culture–negative rate of 16.3% (15). In our previous cohort of patients with culture-negative peritonitis, we postulated that technical problems during the collection of dialysis effluent and/or a history of recent antibiotic therapy could be the underlying reasons (25). Because all patients received prophylactic antibiotic before catheter insertion and because most patients (82.7%) with culture-negative PTP had it occur within 30 days of catheter insertion, we believed that recent antibiotic use was a more important contributing factor. None of the patients with culture-negative peritonitis in the PTP group had evidence of eosinophilic peritonitis defined as presence of turbid PDE, PDE leukocyte count >100/μl, and >10% eosinophils in the PDE differential white cell count.

Polymicrobial peritonitis was the second most common cause of PTP. Although the majority of polymicrobial peritonitis can be successfully treated without catheter removal, the long-term prognosis is poor (26,27). Although traditionally thought to be secondary to underlying bowel pathology, no consistent risk factors have been identified in previous studies (28–30). In the PTP group, six of 11 patients developed polymicrobial peritonitis within 2 weeks of catheter insertion. Inadvertent bowel injury during surgery could not be excluded. Among these six patients, one patient died because of sudden cardiac arrest, one died because of liver failure, and one required catheter removal. The other three patients responded to conservative treatment. These six catheters were inserted by four different nephrologists. For the remaining five patients who developed polymicrobial peritonitis after 2 weeks of catheter insertion, they had undergone IPD uneventfully at least twice before onset of peritonitis. Polymicrobial peritonitis in these patients was considered less likely related to intraoperative bowel injury.

Various risk factors for peritonitis in incident patients on PD have been identified from observational studies (17). We have included the majority of the potential risk factors in our univariate analysis. However, we did not include smoking status, psychosocial factors, and socioeconomic factors, mainly because of unavailability of data. In multivariate analysis, we found that serum albumin was the only independent predictor of PTP. Serum albumin has long been regarded as a marker of morbidity in patients on PD (31). Several studies have shown that serum albumin is also an independent predictor of peritonitis in patients on PD (16,32). Hypoalbuminemia, a marker of malnutrition and inflammation, can also predict all-cause, cardiovascular, and infection-related mortality in patients on PD (33). In our study, we also showed that serum albumin was an independent predictor of mortality. The role of nutritional interventions in patients who are hypoalbuminemic before catheter insertion deserves additional exploration.

We found that patients suffering from PTP had significantly worse patient survival. Early peritonitis has been associated with poor prognosis in patients on PD. In a retrospective study involving 124 patients on PD from Taiwan, it was found that early peritonitis, defined as peritonitis within 20.3 months of PD, was associated with worse patient and technique survival (6). In another recent study involving 155 elderly patients on PD from China, patients suffering from peritonitis within 6 months of PD had significantly worse patient survival than those who had peritonitis after 6 months and those who never had peritonitis (7). However, early peritonitis had no effect on long–term technique survival in this study. Another retrospective study from Canada showed that patients with early peritonitis, defined as having peritonitis within 3 months of PD, had a nearly twofold increased risk of technique failure and death (5). In our study, patients with PTP had significantly lower hemoglobin and serum albumin. These patients were prone to various systemic infections, and infection other than peritonitis accounted for more than one third of mortality. Up to 26.3% of patients with PTP died of termination of dialysis. In fact, among 10 patients in the PTP group who had their PD terminated, seven were elderly patients, and five had termination of PD within 6 months. Also, 15 patients in the control group received a kidney transplant compared with only one

patient in the PTP group. The development of PTP could, indeed, be a reflection of general frailty in these patients.

There are several limitations of our study. This is a single–center, retrospective study, and our results may not extrapolate to other centers. However, this was the first study ever conducted to examine PTP. The sample size was small, mainly because PTP was an uncommon event (4.2% in this cohort). Guidelines suggested that the peritonitis rate after catheter insertion should be <5% (34). Our previous experience showed that peritonitis rate within 1 month of catheter insertion was 3.6% (11). Therefore, we may not have sufficient statistical power to identify certain clinically meaningful differences between the two groups. There could be selection bias, because the PTP group had significantly lower hemoglobin and serum albumin. Because we found that PTP *per se* was not an independent predictor of mortality, the worse patient survival observed in the PTP group could be a result of general frailty as reflected by their lower serum albumin concentration. Initiation of IPD with connections and disconnections could be a risk factor of PTP. In our cohort, however, all patients in both groups required IPD before PD training. This could be related to late catheter insertion when residual renal function was low. Therefore, we could not evaluate whether the performance of IPD was an independent risk factor for peritonitis. We did not have information on *S. aureus* carrier status in our cohort, which could be an important risk factor for *S. aureus* PTP. Our findings, however, serve as an important basis for evaluation of the effect of *S. aureus* carriage on development of PTP. ESI was not uncommon in our study (23.1%), and 83.3% of ESIs were caused by *S. aureus*. The role of *S. aureus* eradication in prevention of PTP is warranted in future studies.

Our study showed, for the first time, that *S. aureus* was the most common causative organism of PTP. Additional studies on the role of universal screening and eradication before catheter insertion are warranted. We observed that serum albumin was the only independent predictor of PTP. Nutritional interventions in patients who are hypoalbuminemic before catheter insertion deserve additional studies.

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Disclosures

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