

Whether to use or Not Prophylactic Antibiotics in Automated Peritoneal Dialysis Patients Undergoing Colonoscopy, A Prospective Controlled Randomized Study

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Abstract

The aim of our study was to look at the overall risk of peritonitis post colonoscopy in end stage renal disease patients on automated peritoneal dialysis and to evaluate the use of prophylactic antibiotic in those patients when given prior to colonoscopy.

A total of 93 patients out of 134 patients on automated peritoneal dialysis (APD) undergoing diagnostic colonoscopy were enrolled in a prospective randomized study. The study extended from January 2016 throughout May 2018 Patients were randomized into two age and sex matched groups; group 1 (46 patients) who had prophylaxis ceftazidime prior to colonoscopy and group 2 (47 patients) who had colonoscopy without prophylactic antibiotics. The following parameters: age, gender, duration on dialysis, duration on APD, diabetic status, use of antibiotics before the procedure, and indications for and findings of colonoscopy were studied. Prophylactic antibiotics were given for prevention of peritonitis if needed according to the 2010 ISPD guidelines.

Results: Post-colonoscopy peritonitis was documented in 2 (4.3%) and 3 (6.4%) patients in groups A and B respectively (p > 0.05). The most common causative agents were gram negative bacteria and there were no other complications.

Conclusion: There was no strong correlation between prophylactic antibiotic use and risk of peritonitis in peritoneal dialysis patients and it seems that the overall risk of developing peritonitis after colonoscopy is low. Only old age, diabetes mellitus and low serum albumin appear to be of significance. Polypectomy; partial or complete did not increase peritonitis episodes in our study population.

Keywords: APD, ESRD, diabetes, colonoscopy, polypectomy, antibiotic prophylaxis, peritonitis.

Introduction

Peritonitis is a well-recognized complication of peritoneal dialysis. The most common causes of peritonitis are probably skin contamination and peritoneal catheter infections. However, the colon is also felt to be a potential source of dialysate contamination, especially in patients who have diverticulitis (1). The incidence of colonoscopy-induced bacteremia is variable, reported from 0% to 27% of patients (2, 3). It has been suggested that antibiotic prophylaxis be given to immunocompromised patients and those with known valvular heart disease or prostheses prior to colonoscopy (4, 5). Few cases have been reported in the literature on peritonitis following colonoscopy in CAPD patients (6-10). These reports suggested that diagnostic instrumental procedures such as colonoscopy may precipitate gram-negative peritoritis in CAPD patients. Due to improvements in peritoneal dialysis technique and the enforcement of the aseptic precautions, there has been a reduction in peritonitis caused by gram-positive but not gram-negative organisms (11). The few case reports in the literature on peritonitis following colonoscopy were all in CAPD but not on APD patients (6, 7, 12-15). In the 2007 series reported by Yip et al (11), 3 of 5 peritonitis episodes were culture negative. The authors could not explain the cause of such a high percentage of culture negative results. The organisms causing these 3 episodes of peritonitis might not have originated from the gastrointestinal tract. In the same series, the risk of developing CAPD peritonitis after colonoscopy in patients without antibiotic prophylaxis was statistically not significant. The 2016 International Society for Peritoneal Dialysis (16) Guidelines showed evidence 2-C favoring the use of prophylaxis antibiotics prior to the procedure (16). However, there has been little literature to support this recommendation. The objective of the present study was to investigate the risks and outcomes of peritonitis after flexible colonoscopy and to show whether there

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is a need for prophylactic antibiotics in automated peritoneal dialysis (APD) patients undergoing this procedure.

Patients and methods

Between January 2016 throughout May 2018, 93 patients, (68 males, 25 females) were included in this study. Patients were randomized (1:1) into two groups; Group A: 46 patients on APD with prophylactic antibiotic therapy before the flexible colonoscopy, Group B: 47 patients on APD without prophylactic antibiotics (Table-1). Exclusion criteria were: history of colonic or rectal resection, neurologic deficit, pregnancy, ongoing sepsis, valvular or chronic heart disease, urinary tract infections, chronic liver disease, exit-site or tunnel infections, pneumonia or pulmonary tuberculosis, peritonitis or history of peritonitis for the last one year and unwillingness to give informed consent (Figure-1). All flexible colonoscopy examinations were performed by trained gastroenterology consultants. All Staff in the endoscopy unit were aware of the potential hazard of cross-infection and assiduous mechanical cleaning followed by disinfection was done. The following parameters: age, gender, duration on dialysis, diabetic state, use of antibiotics before the procedure, and indications for and findings of colonoscopy were studied. APD peritonitis episodes occurring within 1 week after colonoscopy, culture results and outcomes of peritonitis were recorded. At our center, the colonoscopy bowel preparation protocol included a low residue diet 2 days before the examination and patients are instructed to take a fluid diet the day before the procedure. Oral electrolyte lavage solutions or aqueous sodium phosphate solution were used as laxative for bowel preparation. Peritoneal dialysis effluent (PDE) was drained and the patient's abdomen was kept empty before the procedure. Prophylactic antibiotics were given for prevention of peritonitis if needed according to the 2010 ISPD guidelines (17). Prophylactic antibiotics for APD peritonitis prevention were not routine at our center. Peritonitis was diagnosed when abdominal pain and cloudy fluid occurred with or without fever, and when peritoneal fluid white blood cell (WBC) count was >100/mm3, with >50% neutrophils. Episodes with peritoneal eosinophilia but negative bacterial culture were excluded. The PDE was sent for hematological and microbiological examination when patients complained of abdominal pain or if the PDE was turbid. For the microbiological tests, 50 mL peritoneal fluid was centrifuged at 5000g for 15 minutes. The deposit was inoculated on 5% sheep blood agar, MacConkey agar, and Sabouraud agar and incubated aerobically at 35°C for up to 72 hours. All isolates were identified by standard biochemical methods and the identity of the isolates was confirmed using the Vitek Automicrobic System (bioMerieux, Vitek, Hazelwood, Missouri, USA). Antimicrobial susceptibility was tested by the Kirby-Bauer disk diffusion method and results interpreted according to the National Committee for Clinical Laboratory Standards criteria. Reappearance of signs of infection with the same organism(s) isolated in the dialysate within two weeks after the completion of antibiotic treatment was classified as relapse, and not as a new episode.

All Patients were on automated peritoneal dialysis (APD) and their dialytic prescription consisted of 1.36% and 2.27% glucose-based solutions Dianeal® over 9-10 hours night dwell and 7.5% icodextrin (Extraneal®, Baxter Castlebar, Ireland) 2 liters as the last fill for the day dwell. Total daily PD volume ranged between 10-12 liters with a fill volume ranging between 2.0-2.5 liters/cycle.

Colonoscopy procedure

In the procedure room, all patients were given supplemental oxygen (4 L/min) through a nasal cannula, and a 3-lead electrocardiogram, pulse oximetry, and blood pressure were monitored. Only the anesthesiologist certified in advanced life support and who completed a structured training program were permitted to administer propofol under the guidance of the endoscopist. The anesthesiologist who administered the sedative medications and physicians were present for the entire period of sedation and examination. The anesthesiologist attempted to achieve a level of sedation that allowed the patient to tolerate the procedure with minimal to mild pain while maintaining adequate cardiorespiratory function. Propofol induction of sedation was begun with an initial 40-mg bolus (20–30-mg for elderly and smaller patients at the discretion of the endoscopist and anesthesiologist) administered intravenously followed by titration with 10–20-mg boluses. After an initial bolus infusion of propofol, the patient was observed for 30–60 seconds before deciding to administer the next bolus. Fentanyl was administered intravenously in 12.5- or 25-g boluses and midazolam as 0.5–1.0-mg boluses. Additional medication

was titrated at 1–3-minute intervals to achieve or maintain the desired level of sedation. An endoscopy technician was available to assist the colonoscopy with technical maneuvers. This staffing pattern has been used in our endoscopy suite for all sedated procedures for several years and was not changed for the study. The following time points were recorded: initiation of sedation, full sedation (when the nurse and endoscopist mutually agreed the patient was sedated sufficiently to begin the procedure), colonoscope insertion, intubation of the cecum, and colonoscope removal from the anus. Interventional procedures like polypectomy were performed when indicated with disposable polypectomy snare G-Flex. Post polypectomy bleeding (if any) was managed by epinephrine injection, hemoclip and heat probe.

Biopsies were taken when indicated by disposable biopsy forceps (Endow by Olympus). After the procedure, both the physician and the nurse completed a questionnaire that assessed the patient's level of sedation, pain, and ability to cooperate. Any complications (decline in oxygen saturation to less than 85%, heart rate less than 50 beats per minute, blood pressure less than 90/50 mm Hg, or need for mechanical ventilation) were recorded.

Prophylactic antibiotic therapy

Antibiotic prophylaxis in our center consisted of first-line antibiotic regimen for APD peritonitis was first- or second-generation cephalosporin plus an aminoglycoside, either tobramycin or netilmicin. Cefazolin combined with ceftazidime was also used as alternative.

Peritonitis therapy

Peritonitis episodes were treated with our center's standard antibiotic protocol, which has been changed systematically over time. The first-line antibiotic regimen for APD peritonitis was first- or second-generation cephalosporin plus gentamicin (loading dose 60 mg i.v. + 4-5 mg/L intraperitoneal). Cefazolin or cefoxitin (2 g i.v. + 50 mg/L intraperitoneal) combined with ceftazidime (2 g i.v. + 1 g intraperitoneal) was also used in our PD unit since the year 2010 according to the ISPD peritonitis guidelines (17). Vancomycin was used as a second-line therapy for primary nonresponding patients. Antibiotic regimens for individual patients were modified when culture results became available. Treatment usually lasted for either 2 weeks or at least 7 more days after normalization of the effluent WBC count, whichever was longer. Requirement of cessation of peritoneal dialysis, temporarily or permanently, and death during peritonitis, were defined as treatment failure. Heparin administration (500-1000 IU/L of dialysis fluid) and exchange of tubing was performed routinely in all cases of peritonitis. The indications for catheter removal included peritonitis caused by *Pseudomonas species*, peritonitis caused by fungi, cases with prolonged course or multiple recurrences, and episodes with suspected bowel perforation.

Statistical methods

Continuous variables are expressed as mean \pm SD and categorical variables are expressed as percentage. Non parametric Spearman Rank test was used for continuous variables correlation and Mann-Whitney test used for comparison of two groups. P values were not adjusted for multiple testing and therefore should be considered descriptive. Variables with significant univariate associations were candidates for multivariate analysis. Univariate and multivariate analysis was used to study the relationship of age, sex, diabetes mellitus, time on APD, hemoglobin and albumin levels and prophylactic antibiotic use with post-colonoscopy peritonitis. The statistical analyses were limited to data regarding only the first episode of peritonitis, unless otherwise noted. Statistical significance was accepted at p < 0.05. The statistical analysis was performed using SPSS for Windows version 20 (IBM Inc. New York, USA).

Results

This prospective randomized study of patients with ESRD on APD and undergoing colonoscopy was performed according to *The Declaration of Helsinki* at King Fahd University Hospital, Al-Khobar, Saudi Arabia. The study was conducted from March 2012 throughout April 2016 with prior approval by *King Fahd Hospital Human Ethical committee*. All patients were above 18 years of age and written

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informed consents were obtained from every patient after full explanation of the aim of the study, the complications of colonoscopy and the expected outcomes. Pregnant females, patients with ongoing sepsis, valvular or chronic heart disease, urinary tract infections, chronic liver disease, exit-site or tunnel infections, pneumonia or pulmonary tuberculosis, peritonitis or history of peritonitis for the last one year were excluded from the study (Figure-1). In a total of 134 APD patients included during the study period of 4 years, 96 colonoscopies were performed in 93 APD patients. Indications for repeating the procedure were inadequate preparation in one and partial or incomplete resection of a sessile polyp in two patients. Mean age was 62.3 ± 9.4 years and duration of dialysis was 35.2 ± 10.6 months; 34 (36.6%) patients were diabetics. The 93 APD patients included in the study were randomized into two groups; group-A (46 patients) who received ceftazidime prophylaxis prior to colonoscopy and group-B (47 patients) who had colonoscopy without antibiotic prophylaxis. Randomization was 1:1. Demographic characteristics of patients are summarized in table-1. The two groups were age and sex matching. Diabetes mellitus was present in 34.8% and 38.3% and hypertension in 82.6% and 76.6% in the two groups respectively (p > 0.05). Mean duration of diabetes mellitus and the duration on APD was 18.8 + 10.7 years and 20.5 + 10.2 years, 31.1 + 11.8 months and 30.6 + 12.5 months in the groups A and B respectively (p > 0.05). The difference in overall fasting blood sugar (FBS) and hemoglobin A1-C (Hgb A1-C) was not statistically significant between the two groups (p > 0.05). At the time of colonoscopy, the mean blood urea nitrogen (BUN), serum creatinine and renal creatinine clearance were 44.18 + 10.23 mg/dl and 46.12 + 9.81 mg/dl; 8.28 + 2.55 mg/dl and 8.33 + 1.87 mg/dl; 6.3 + 2.1 and 6.1 + 2.8 ml/min in groups A and B respectively (p > 0.05). Mean hemoglobin level, serum potassium (K+) and serum albumin were similar in both groups at the time of the procedure (p > 0.05) (table-1). Indications for and findings of colonoscopy are summarized in table-2 and figure-2. Of all colonoscopies 60.2% showed normal findings, 17.2% with colonic polyps at different sites, 12.9% with angiodysplastic-like lesions, 5.4% with colonic ulcer (s), 3.2% with diverticula without diverticulitis and 1.1% had transverse colon stricture which was managed with stent insertion. Inflammatory bowel disease in the three patients was inactive for more than one year. Findings at colonoscopy are shown in figure-2. Post-colonoscopy peritonitis was documented in 2 (4.3%) and 3 (6.4%) patients in groups A and B respectively (p > 0.05); the causative organisms were mainly gram-negative bacteria (4 out of 5 cases were gram negative bacteria and one with Candida albicans) (table-3). Peritonitis episodes were not documented in any patient with diverticulosis or biopsied colonic polyps. All peritonitis cases resolved with treatment and one of the patients in group A required catheter removal because of fungal peritonitis. Complications other than peritonitis were 0.0% in both groups. Different variables were analyzed to demonstrate its relation with peritonitis episodes (Table-4). No significant difference in serum BUN or serum creatinine was observed between those who developed peritonitis and those who did not in the two groups (p > 0.05). Seven factors met the criteria for inclusion in the univariate analysis: age (≥ 60) (odds ratio [OR]=1.41, 95% confidence interval [95% CI]=1.11–1.6, P=0.0336), male sex (OR=0.79, 95% CI= 0.66-0.93, P= 0.0462), diabetes mellitus (OR=1.23, 95% CI=1.15-1.62, P,0.0389), duration on APD (OR=1.58, 95% CI=1.24-1.81, P=0.0308), hemoglobin level (OR=0.89, 95% CI=0.83-1.1, P= 0.0430), prophylactic antibiotics (OR=1.18, 95% CI=0.92-1.15, P=0.0481), and serum albumin (OR=2.24, 95% CI=1.98-2.66, P=0.0292). With multivariate analysis Only age (OR=1.34, 95% CI=1.16–1.64, P=0.0326), diabetes mellitus (OR=0.79, 95% CI=0.68-0.11, P=0.0279) and albumin levels (OR= 0.84, 95% CI= 1.12-1.36, P= 0.2253) were associated significantly with post colonoscopy peritonitis.

Discussion

Peritonitis remains the most serious complication of peritoneal dialysis. Around 18% of the infection-related mortality in PD patients is the result of peritonitis. Although less than 4% of peritonitis episodes result in death, peritonitis continues to be a leading factor to death in 16% of deaths on PD (18). In addition, peritonitis is probably the most common cause of technique failure in PD, and it remains a major cause of patients discontinuing PD and switching to hemodialysis. Therefore, the PD community continues to focus attention on prevention and treatment of PD-related infections (18-26). Peritonitis caused by enteral micro -organisms is relatively infrequent in PD patients (27-29). The source of contamination in those cases not associated with catheter exit-site or tunnel infections is thought to be

transmural (1, 27). Micro-organisms can gain access to the peritoneum from the intestinal lumen or through genital organs (30, 31). Diagnostic instrumental procedures, such as colonoscopy, have been implicated in the development of these peritonitis episodes (14, 15). However, in many cases there is no evidence that links peritonitis to colonoscopy as a risk factor (29, 30). The recommendations concerned with colonoscopy in PD patients are not based solely on randomized controlled trials because such studies in PD patients are limited, where there is no definitive evidence but the group feels there is sufficient experience to suggest a certain approach, this is indicated as "opinion" based. The recommendations are not meant to be implemented in every situation but are recommendations only. Each center should examine its own pattern of infection, causative organisms, and sensitivities and adapt the protocols as necessary for local conditions (19). Post colonoscopy peritonitis in patients undergoing PD can result from translocation microorganisms across the bowel wall (32) and it has been alleged that gastrointestinal endoscopic procedures in those patients can lead to peritonitis (33). Contrary to Yip et al who, in a selected cohort of PD patients with indications of colonic examinations, suggested that diverticulosis, may be a risk factor for the development of enteric peritonitis, we did not encounter peritonitis episodes in our 3 diverticulosis patients. Colonic diverticulosis did not appear to affect the outcome of colonoscopy in our patients. Supporting our findings was the report by Toda et al. (34) who studied 317 PD-candidate patients over approximately 4 years and concluded that asymptomatic diverticulosis identified by computed tomography was not a risk factor for enteric peritonitis in their study population. A retrospective study by Tip et al. (35) found that the risk of peritonitis after colonoscopy without antibiotic prophylaxis was 6.3%. The authors however, indicated that it lacks statistical significance compared with prior antibiotic prophylaxis (35). Colon biopsy or polypectomy did not appear to further increase the risk of peritonitis in our cohort (11). Interestingly, the International Society for Peritoneal Dialysis recommended antibiotic prophylaxis before any procedure involving the abdomen or pelvis, including colonoscopy (16). Again, it is important to notice that these recommendations were based only on observational studies and case reports. The 2005 and the 2016 ISPD guidelines suggested empirical 1-gram ampicillin or aminoglycoside with or without metronidazole before colonoscopy (16, 36). These guidelines recommend antibiotic prophylaxis for CAPD patients undergoing colonoscopy with polypectomy; however, there has been little literature to support these recommendations. Studies on these guidelines are rare, and randomized controlled studies to support this recommendation are lacking. Moreover, these new guidelines clearly stated that the optimal antibiotic regimen has not been determined by clinical study yet (16). Similar suggestions were made by the Dutch Federation of Nephrology with the addition of dialysate drainage before the procedure (37). However, these suggestions have not gained wide acceptance. Contrary to the suggestions above, the American Society for Gastrointestinal Endoscopy and the British Society of Gastroenterology do not suggest prophylactic antibiotics before colonoscopy (38, 39). There exists a lack of consensus on this issue. There have been few case reports in the literature on peritonitis following colonoscopy in peritoneal dialysis patients (6, 7, 14-17). These reports suggested that instrumental procedures such as colonoscopy may precipitate gram-negative peritonitis in PD patients. On the other hand, some literature reported bacterial peritonitis following endoscopic polypectomy in peritoneal dialysis patients despite antibiotics prophylaxis (10). So far there are no strong data demonstrating a causal association between endoscopic procedures and bacteremia or that antibiotic prophylaxis prior to endoscopic procedures protects against bacteremia. Much of the existing data reflects estimated risk associated with conventional endoscopic techniques. There are no results available that confidently quantify bacteremia rates with newer endoscopic procedures such as per oral endoscopic myotomy, endoscopic submucosal dissection, flexible colonoscopy or polypectomy (11). We studied APD patients with and without antibiotic prophylaxis before flexible sigmoidoscopy. The difference in peritonitis episodes in our study between the two groups was not statistically significant (4.3% vs. 6.4%, p > 0.05). Surprisingly, none of the post-polypectomy and none of our patients with diverticular disease (without diverticulitis) had post-procedure peritonitis in our cohort; finding that correlates with the report of Yip, et al. (11) who stated that colonic biopsy or polypectomy was not associated with a higher risk of peritonitis in their CAPD patients. In addition, we did not encounter serious complications of colonoscopy i.e. perforation or hemorrhage. Transient bacteremia occurs frequently during routine daily activity, often at rates exceeding those associated with endoscopic

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procedures. Brushing and flossing of teeth has been associated with rates of bacteremia of 20% to 68%, use of toothpicks with rates of 20% to 40%, and even activity that might be considered entirely physiologic, such as chewing food, with rates ranging from 7% to 51% (40). By multivariate analysis, the use of prophylactic antibiotics prior to colonoscopy was not a predictive variable for developing peritonitis in our study population, on the contrary, other factors namely age, diabetes mellitus and serum albumin levels proved to be significant predictive variables for post-colonoscopy peritonitis. One patient from the group of those who received prophylactic antibiotics had Candida species in peritoneal fluid culture. Although we could not prove the relation between antibiotic prophylaxis and the development of this un-expected growth, it is not unreasonable to speculate that antibiotic administration may have favored intestinal non-bacterial overgrowth (Candida in our case) which, potentially, may have conditioned the pathogenicity of these organisms in that patient. The human colonic microflora ecosystem, its metabolic functions, and its colonization resistance are vital for the well-being of the host, production of vital metabolites, and prevention of infection. In a study by Edlund and Nord (41) marked ecological disturbances were seen in the intestinal microflora during antibiotic treatment. The numbers of enterococci, enterobacteria (except E. coli) and peptostreptococcus increased significantly during treatment. Eight patients became newly colonized by Klebsiella spp. and Citrobacter freundii during treatment. The number of patients colonized with yeasts (mostly C. albicans) increased from zero to nine during treatment; two patients were still colonized with yeasts after treatment. Sullivan et al (42, 43) reported that administration of antimicrobial agents, therapeutically or as prophylaxis, causes disturbances in the ecological balance between the host and the normal intestinal microflora and that by using antimicrobial agents, the risk of emergence and spread of resistant strains between patients and dissemination of resistant micro-organisms increases significantly. In a study concerned with a similar matter, Berg (44) concluded that the colonic microflora appears to stimulate the host immune system to respond rapidly to pathogen challenges. Although the cells of the intestinal tract coexist with the normal commensal microflora, they recognize and clear invading pathogens before returning to homeostasis with the commensal bacteria. The colonic microflora provides a number of benefits, including contributing to the host's nutrition and protecting the host from infection. In most cases of antimicrobial prophylaxis or therapy, the bacterial populations in some genera are reduced in numbers while those in other genera increase. In some cases, the increased numbers of certain bacteria are accompanied by resistant strains of bacteria or overgrowth by fungi. Treatment with antimicrobial combinations does not necessarily prevent resistance development. It may even result in fungal overgrowth and appearance of bacteria with resistance to all of the drugs in the combination (45). Given the notorious possibility of resistant strains' development and the relative rarity with which most PD patients undergo colonoscopy procedures, the frequency and risk of colonoscopy-related bacteremia, as we demonstrated in our study, is trivial compared with the frequency of bacteremia encountered with routine daily activity. This provides a strong rationale against routine administration of antibiotic prophylaxis prior to all endoscopic procedures.

There are some limitations, however, in our study. First, this study was conducted in a single tertiary medical center, and endoscopy-associated complications may vary in different hospitals. Second, the study was conducted on a selected group of APD patients after applying strict exclusion criteria. Third, the study recorded only 93 endoscopic procedures and may have underestimated the importance of antibiotic prophylaxis. Therefore, larger randomized trials are required to explore the necessity of antibiotic prophylaxis in the prevention of postcoloscopy PD peritonitis. Nevertheless, our study has the strength of being the first prospective randomized study in this field.

Conclusion

The relation between prophylactic antibiotic use prior to colonoscopy in APD patients was lacking and the overall risk of peritonitis in general is low in this population. Only old age, diabetes mellitus and low serum albumin appeared to be of significance. Neither polypectomy; partial or complete nor diverticulosis were associated with increased incidence of post-colonoscopy peritonitis. The study, however, the study recorded limited number of patients and may have underestimated the importance of antibiotic prophylaxis. Therefore, larger prospective randomized trials are needed.

Table 1. Demographic characteristics of the study population

	Group A	Group B	p
	(n = 46)	(n = 47)	-
Age (years), mean <u>+</u> SD	61 <u>+</u> 12.5	63 <u>+</u> 11.8	> 0.05
Female/Male (female %)	11/35 (31.4)	13/34 (38.2)	> 0.05
Smokers (%)	21.7	19.1	> 0.05
Hypertension, n (%)	38 (82.6)	36 (76.6)	> 0.05
BMI at beginning, mean ±	29.1 <u>+</u> 4.1	29.3 <u>+</u> 3.8	> 0.05
SD			
Diabetes mellitus, n (%)	16 (34.8)	18 (38.3)	> 0.05
Duration of diabetes,	18.8 <u>+</u> 10.7	20.5 <u>+</u> 10.2	> 0.05
(years), mean <u>+</u> SD			
Duration on APD, months	31.1 <u>+</u> 11.8	30.6 <u>+</u> 12.5	> 0.05
$(\text{mean} \pm \text{SD})$			
Overall FBS in diabetics,	8.6 <u>+</u> 1.3	8.4 <u>+</u> 1.4	> 0.05
mmol/L (mean + SD)			
Overall Hgb A1C % in	$7.1\% \pm 0.5$	6.8 ± 0.8 .	> 0.05
diabetics (mean \pm SD)			
Hgb at colonoscopy, gm/dl	10.16 ± 2.25	10.32 <u>+</u> 2.77	> 0.05
$(\text{mean} \pm \text{SD})$			
BUN at colonoscopy, mg/dl	44.18 <u>+</u>	46.12 <u>+</u> 9.81	> 0.05
$(\text{mean} \pm \text{SD})$	10.23		
Serum Cr. at colonoscopy,	8.28 <u>+</u> 2.55	8.33 <u>+</u> 1.87	> 0.05
mg/dl (mean + SD)			
Serum K+ (mEq/L)	4.1 <u>+</u> 1.9	3.9 <u>+</u> 2.1	> 0.05
Serum albumin (gm/l)	3.8 <u>+</u> 2.0	3.7 <u>+</u> 1.8	> 0.05
Renal Cr Cl. ml/m (mean +	6.3 <u>+</u> 2.1	6.1 <u>+</u> 2.8	> 0.05
SD)			

BMI: Body mass index, APD: automated peritoneal dialysis, FBS: Fasting blood sugar, Hgb: hemoglobin, BUN: blood urea nitrogen, Cr: creatinine, K+: potassium, Cr Cl: creatinine clearance.

Table 2. Indications for and findings of colonoscopy

Number (%)	Indication	Findings (number)	Action (number)
17 (18.3)	Screening for colonic	Normal (13)	None (13)
	Cancer	Transverse and descending	Biopsies and removal
		colon polyps (4)	(4)
15 (16.1)	Investigation for iron	Normal (11)	None (11)
	deficiency anemia	Angiodysplastic like lesions	Biopsies & bleeding
		(4)	protocol (4)
14 (15.1)	Altered bowel habits	Normal (9)	None (9)
	(chronic diarrhea or	Diverticulae (3)	None (3)
	chronic constipation)	Transverse colon polyps (2)	Biopsies and removal
			(2)
12 (12.9)	Positive fecal occult	Normal (5)	None (5)
	blood testing without	Angiodysplastic-like lesions	Biopsies & bleeding
	overt rectal bleeding	(4)	protocol (4)
		Descending colon polyp (3)	Biopsies and removal
			(3)
9 (9.7)	Overt rectal bleeding	Normal (1)	None (1)
		Transverse or descending	Biopsies & bleeding
		colon ulcers (2)	protocol (2)

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		Angiodysplastic-like lesions (3) Ascending & transverse colon	Biopsies & bleeding protocol (3) Biopsies and removal
		polyp (3)	(3)
8 (8.6)	Finding of polyp (s)	Normal (5)	None (5)
	during	Descending colon polyps (2)	Biopsies and removal
	sigmoidoscopy	Angiodysplastic-like lesions	(2)
		(1)	Biopsies & bleeding protocol (1)
8 (8.6)	Bloody effluent	Normal (7)	None (7)
		Transverse colon polyp (1)	Biopsies and removal
			(1)
7 (7.5)	Family history of	Normal (5)	None (5)
	colon cancer or	Ascending colon polyp (1)	Biopsies and removal
	polyps	Descending colon ulcer (1)	(1)
			Biopsies (1)
3 (3.2)	Inflammatory bowel	Transverse and/or descending	Biopsies (2)
	disease	colon ulcers (2)	Stent (1)
		Transverse colon stricture (1)	

Table 3. Microorganisms responsible for peritonitis

Patient's No#	Group A (2 cases) Microorganisms	Patient's No#	Group B (3 cases) Microorganisms
12	E. coli + Enterococcus faecalis	5	E. coli
34	Candida albicans	22	Klebsiella species
		40	Enterococcus

Table 4. Comparison of characteristics of patients with and without peritonitis after colonoscopy

	Group A	p	Group B	p
	Peritonitis No peritonitis		Peritonitis No	
			peritonitis	
Number (%)	2 (4.3) 44 (95.7)		3 (6.4) 44 (93.6)	0.1742
Age (year)	66.5 ± 10.8 54.3 ± 11.2	0.0370	65.1 <u>+</u> 12.1 55.2 <u>+</u>	0.0342
			10.5	
Sex (M/F)	3/0 32/12	0.0461	3/1 30/12	0.0432
Diabetes, n (%)	2/2 (100) 14/44 (31.8)	0.0412	3/3 (100) 15/44	0.0358
			(34.1)	
Duration on APD,	33.4 <u>+</u> 9.6 21.1 <u>+</u> 11.3	0.0281	31.6 <u>+</u> 7.7 23.5 <u>+</u>	0.0320
month, (mean)			9.2	
BUN, mg/dl	44.8 ± 10.7 43.3 ± 11.2	0.5804	46.4 <u>+</u> 8.8 44.8 <u>+</u> 9.1	0.6213
(mean)				
Creatinine, mg/dl	8.5 ± 2.2 8.8 ± 2.5	0.4036	$7.8 \pm 2.69.0 \pm 2.1$	0.3892
(mean)				
Hemoglobin, gm/dl	7.5 <u>+</u> 3.4 9.6 <u>+</u> 2.2	0.0447	$7.4 \pm 2.7 \ 10.1 \pm 1.4$	0.0355
(mean)				
Serum K+, mEq/l	$3.8 \pm 2.1 \ 3.8 \pm 1.8$	0.4982	$4.0 \pm 2.3 \ 3.7 \pm 2.6$	0.4432
(mean)				
Serum albumin,	$2.3 \pm 2.0 \ 4.3 \pm 0.8$	0.0283	2.4 <u>+</u> 1.9 4.1 <u>+</u> 1.2	0.0311
gm/dl (mean)				

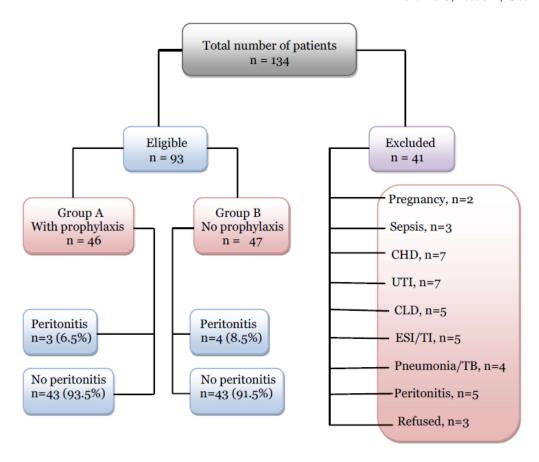


Figure 1. Consort diagram demonstrating study design and patients' progress.

CHD: chronic or valvular heart disease, UTI: urinary tract infection, CLD: chronic liver disease, peritonitis: ongoing or previous.

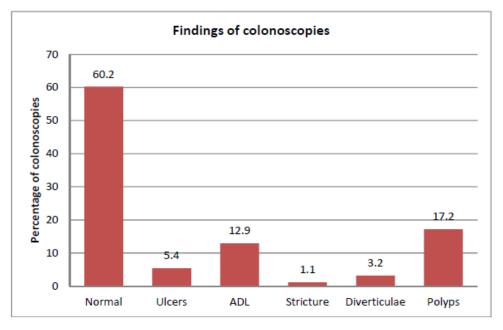


Figure-2. Finding of colonoscopies in the study population

ADL: Angiodysplastic-like lesions

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References

[1].Allison MC, Sandoe JA, Tighe R, Simpson IA, Hall RJ, Elliott TS; Endoscopy Committee of the British Society of Gastroenterology: Antibiotic prophylaxis in gastrointestinal endoscopy. Gut 2009; 58: 869-80.

- [2].ASGE Standards of Practice Committee, Banerjee S, Shen B, Baron TH, Nelson DB, Anderson MA, Cash BD, Dominitz JA, Gan SI, Harrison ME, Ikenberry SO, Jagannath SB, Lichtenstein D, Fanelli RD, Lee K, van Guilder T, Stewart LE: Antibiotic prophylaxis for GI endoscopy. ASGE guidelines. Gastrointest Endosc 2008; 67: 791-98.
- [3].Bac DJ, van Blankenstein M, de Marie S, Fieren MW. Peritonitis following endoscopic polypectomy in a peritoneal dialysis patient: the need for antibiotic prophylaxis. Infection 1994; 22:220–1.
- [4].Bayston R, Andrews M, Rigg K, Shelton A. Recurrent infection and catheter loss in patients on continuous ambulatory peritoneal dialysis. Perit Dial Int 1999; 19:550–55.
- [5].Bunke CM, Brier ME, Golper TA. Outcomes of single organism peritonitis in peritoneal dialysis: gramnegatives versus gram-positives in the Network 9 Peritonitis Study. Kidney Int 1997; 52:524–29.
- [6].Choi P, Nemati E, Banerjee A, Preston E, Levy J, Brown E. Peritoneal dialysis catheter removal for acute peritonitis: a retrospective analysis of factors associated with catheter removal and prolonged postoperative hospitalization. Am J Kidney Dis 2004; 43:103–11.
- [7].Coward RA, Gokal R, Wise M, Mallick NP, Warrell D. Peritonitis associated with vaginal leakage of dialysis fluid in continuous ambulatory peritoneal dialysis. Br Med J 1982; 284:1529.
- [8].Edlund C, Nord CE. 1999b. Effect of quinolones on intestinal ecology. Drugs, 58(Suppl 2):65–70.
- [9].Fatemeh Rafii, John B Sutherl and, Carl E Cerniglia. Effects of treatment with antimicrobial agents on the human colonic microflora. Therapeutics and Clinical Risk Management 2008:4(6) 1343–57.
- [10]. Fried L, Bernardini J, Piraino B. Iatrogenic peritonitis: the need for prophylaxis. Perit Dial Int 2000; 20: 343-345
- [11]. Johnson DW, Dent H, Hawley CM, McDonald SP, Rosman JB, Brown FG, et al. Associations of dialysis modality and infectious mortality in incident dialysis patients in Australia and New Zealand. Am J Kidney Dis 2009; 53:290–97.
- [12]. Katsanos KH, Tsianos EV. Bacterial peritonitis following multiple endoscopic polypectomy in a peritoneal dialysis patient despite antibiotics prophylaxis. Annals of Gastroenterology 2010; 23: 211-212.
- [13]. Kumar S, Abcarian H, Prasad L, *et al.* Bacteremia as sociated with lower gastrointestinal endoscopy, fact or fiction? *Dis Colon Rectum.* 1982; 25:131-134.
- [14]. Macrae F, Tan K, Williams C. Towards safer colonoscopy: A report on the complications of 5000 diagnostic or therapeutic colonoscopies. *Gut.* 1983: 24:376-383.
- [15]. Oreopoulos DG. Prevention of peritonitis in patients undergoing CAPD. Perit Dial Bull. 1986; 6:2-3.
- [16]. Petersen JH, Weesner RE, Giannella RA. Escherichia coli peritonitis after left-sided colonoscopy in a patient on continuous ambulatory peritoneal dialysis. Am J Gastroenterol 1987; 82:171–2.
- [17]. Philip Kam-Tao Li, Cheuk Chun Szeto, Beth Piraino, Judith Bernardini, Ana E. Figueiredo, Amit Gupta, David W. Johnson, Ed J. Kuijper, Wai-Choong Lye, William Salzer, Franz Schaefer, and Dirk G. Struijk10. Peritoneal dialysis-related infections recommendations: 2010 update. Perit Dial Int, 30: 393–423.
- [18]. Philip Kam-Tao Li, Cheuk Chun Szeto, Beth Piraino, Javier de Arteaga, Stanley Fan, Ana E. Figueiredo, Douglas N. Fish, Eric Goffin, Yong-Lim Kim, William Salzer, Dirk G. Struijk, Isaac Teitelbaum, and David W. Johnson. ISPD peritonitis recommendations: 2016 update on prevention and treatment. Perit Dial Int, in Press www.PDIConnect.com
- [19]. Piraino B, Bernardini J, Sorkin M. The influence of peritoneal catheter exit-site infections on peritonitis, tunnel infections, and catheter loss in patients on continuous ambulatory peritoneal dialysis. Am J Kidney Dis 1986; 8:436–40.
- [20]. Piraino B, Bernardini J, Sorkin M. Catheter infections as a factor in the transfer of continuous ambulatory peritoneal dialysis patients to hemodialysis. Am J Kidney Dis 1989; 13:365–69.
- [21]. Piraino B, Bernardini J, Brown E, et al. ISPD position statement on reducing the risks of peritoneal dialysis—related infections. Perit Dial Int 2011; 31:614-30.
- [22]. Poortvliet W, Selten HP, Raasveld MH, et al. CAPD peritonitis after colonoscopy: follow the guidelines. Neth J Med 2010; 68:377-78.
- [23]. Sipahi S, Gungor O, Kircelli F, Aydin B, Ulker EA, Tamer A. Peritonitis after colonoscopy in a peritoneal dialysis patient. Turk Nephrol Dial Transpl 2012; 21 (1): 105-106.

- [24]. Sullivan A, Edlund C, Nord CE. 2001a. Effect of antimicrobial agents on the ecological balance of human microfl ora. Lancet Infect Dis, 1:101–14.
- [25]. Sullivan A, Edlund C, Svenungsson B, et al. 2001b. Effect of perorally administered pivmecillinam on the normal oropharyngeal, intestinal and skin microfl ora. J Chemother, 13:299–308.
- [26]. Szeto CC, Chow KM, Wong TY, Leung CB, Li PK. Conservative management of polymicrobial peritonitis complicating peritoneal dialysis—a series of 140 consecutive cases. Am J Med 2002; 113:728–33.
- [27]. Terence Yip, Kai Chung Tse, Man Fai Lam, Suk Wai Cheng, Sing Leung Lui, Sydney Tang, Matthew Ng, Tak Mao Chan, Kar Neng Lai, and Wai Kei Lo. Risks and outcomes of peritonitis after flexible colonoscopy in CAPD patients. Perit Dial Int 2007; 27:560–564.
- [28]. Verger C, Danne O, Vuillemin F. Colonoscopy and continuous ambulatory peritoneal dialysis. Gastrointest Endosc 1987; 33:334–5.
- [29]. Wilson W, Taubert KA, Gewitz M, et al. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes
- [30]. Research Interdisciplinary Working Group. Circulation 2007; 116:1736-54.
- [31]. Woodrow G, Turney JH, Brownjohn AM. Technique failure in peritoneal dialysis and its impact on patient survival. Perit Dial Int 1997; 17:360–4.
- [32]. Yip T, Tse KC, Lam MF, et al. Colonic diverticulosis as a risk factor for peritonitis in Chinese peritoneal dialysis patients. Perit Dial Int 2010; 30:187-91.