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CLINICAL STUDY

The Prevalence and Clinical Features of Tuberculous Peritonitis in CAPD Patients in Turkey, Report of Ten Cases from Multi-centers

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ABSTRACT

Objective. To determine the rate, risk factors and outcome of Tuberculous Peritonitis (TBP) in patients treated with continuous ambulatory peritoneal dialysis (CAPD) in our units. *Design*. Retrospectively, we reviewed the medical

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data of all CAPD patients from 12 centers for TBP, covering the period between 1986 and December 2002. *Setting*. All patients were from 12 renal clinics at tertiary-care university hospitals. *Results*. Ten cases of TBP were identified among the CAPD patients in our centers. There were five male and five female patients with a mean age of 37.2 years. None of the patients had tuberculosis history, 6 patients had predominance of PNL. One patient had coincidental bacterial peritonitis. Two patients were successfully treated without the removal of the Tenckhoff catheter. *Conclusion*. TBP in CAPD patients is a very rare complication. In contrast to predominance of lymphocytes in nonuremic patients with tuberculous peritonitis, CAPD patients with tuberculous peritonitis may have predominance of PNL on examination of the peritoneal fluid. Since TBP has high morbidity and mortality, early diagnosis and treatment of disease are extremely important for improving outcome.

Key Words: CAPD; Tuberculous peritonitis; Prevalence; Outcome; Predominance of PNL.

INTRODUCTION

Peritonitis has been the most common major clinical problem and is often the cause for failure of continuous ambulatory peritoneal dialysis (CAPD).[1] Gram-positive microorganisms can be easily identified as a common cause of peritonitis. Gram-negative microorganisms can also be a cause of peritonitis in some patients.[1,2] Anaerobic bacteria, fungi, and mycobacteria account for less than 5% of the peritonitis complicating CAPD.[3] In Turkey, although the incidence of peritonitis varies from center to center, it ranges from 1 episode in every 22 to 50 patient treatment months (mean 33 months).[4] It has progressively declined for the last two decades due to better patient education, and improvement in new connector and catheter technologies for CAPD.

Although pulmonary and extrapulmonary tuberculosis is an infection with higher incidence in patients with chronic renal failure, tuberculosis peritonitis (TBP) in patients with CAPD has been described rarely .[5–24] TBP is rare, but it carries a high risk for morbidity, mortality, and treatment failure .[23] Early diagnosis is essential. The mortality rate is almost 25% at 9 months after diagnosis.[23]

Tuberculous peritonitis in patients with CAPD has been reported in 1–2% of the peritonitis cases.[25,26] The treatment and outcomes of TBP in patients with CAPD have not been well described in literature yet. In this multicenter study, we report the rate, risk factors, and our experience in treatment of TBP with antituberculous drugs (ATDs).

PATIENTS AND METHODS

The medical archives of all CAPD patients from twelve centers were analyzed for tuberculous peritonitis retrospectively, covering the period between March 1986 and December 2002. The onsets of peritonitis, clinical courses, use of ATDs, catheter

removal and medical predisposing factors have been evaluated. The patients with positive Acid-Fast Bacilli (AFB) smear or culture from peritoneal dialysis fluid (PDF) were identified as TBP.

All of the patients with culture-negative peritonitis and unresponsive to conventional peritonitis therapy, were also evaluated for TBP. Smear and culture for AFB from PDF were routinely examined in the patients with culture negative peritonitis. Using BACTEC Methods performed culture of mycobacterium tuberculosis from PDF.

The onset of peritonitis was defined as the time when symptoms and signs of peritonitis were presented. The diagnosis for TBP was established by the presence of positive smear or culture for AFP from PDF. White blood counts (WBC) in PDF were also differentiated into PNL and lymphocytes. Response to treatment was defined as the resolution of clinical signs and symptoms of peritonitis and the normalization of PDF WBC count. The short-term outcome of TBP was evaluated.

RESULTS

Ten cases of TBP were identified among 2004 patients in our centers. The prevalence of TBP was approximately 0.5%. The clinical features of the patients are shown in Table 1. There were five male and five females with mean age of 37.2 years (range 20 to 49 years). Duration of CAPD treatment before the onset of TBP was 13.2 months (range 5–40 months). One patient (patient no. 8) had received immunosuppressive medications. None of the patients had tuberculosis history. Two patients had diabetes. Two of the patients had radiological finding of tuberculosis (patients no. 3 and 8). All patients were on CAPD treatment when the TBP was diagnosed.

The PDF characteristics are shown in Table 2. All patients had abdominal pain and cloudy fluids. Five patients had also fever. Only one patient had radiological evidence of tuberculosis infection on thorax-computed tomography (Case no. 8). One patient (patient no. 3) had pleural effusion on chest X ray. Total PDF WBC counts ranged from 300 to 5000 WBC/mm^3. Three patients had predominant lymphocytosis (72%, 74%, 82% respectively), 6 patients had predominant PNL (range 80–90%). One patient had coincidental bacterial peritonitis.

The results of diagnostic methods are outlined in Table 3. Positive smear for AFD was present in 4, positive culture for mycobacterium tuberculosis in 6; positive histopathological findings for tuberculous from peritoneal biopsy in 2 patients. The average interval between onset of peritonitis and diagnosis was 2.7 months (range 1–8 months). Before diagnosis of TBP, all patients were on regular peritonitis treatment as shown in Table 4. In one patients (patients no. 2) fluconazole was started due to failing to respond to antibiotics. After the diagnosis of TBP was established, ATDs were initiated in all patients.

Once the diagnosis of TBP was made the patients were placed on isoniazid, rifampicin, morphazinamide, pyrazinamide, or ethambutol at the conventional doses. Combination therapy of 3–4 ATDs was used in unmodified doses in all patients including isoniazid and rifampicin. Two patients also received ciprofloxacin. Treatment protocols and short-term outcomes of the patients are summarized in Table 3.

Table 1. Characteristics of the patients with TBP undergoing CAPD.

Patient no.	Age/ sex	Cause of renal failure	Duration of treatment of CAPD (months)	Predisposing medical conditions	Fever	Abdominal pain
1	42/F	Unknown	19	−	−	+
2	20/M	Membranous glomerulonephritis	5	Chronic hepatitis	−	+
3	31/M	Hypertension	12	−	−	+
4	49/M	Diabetic nephropathy	29	Diabetes mellitus	−	+
5	45/F	Hypertension	7	−	+	+
6	49/M	Diabetic nephropathy	9	Diabetes mellitus	−	+
7	36/F	Chronic glomerulonephritis	2	−	+	+
8	25/F	Membrano-proliferative glomerulonephritis	3	Recent immuno suppression, chronic hepatitis	+	+
9	45/M	Hypertension	6	−	+	+
10	30/F	Chronic glomerulonephritis	40	−	+	+

Table 2. Clinical and peritoneal fluids characteristics in the patients with TBP.

	Clinical features				Peritoneal-fluid characteristics				
Patients no.	Cloudy fluid	Coincident bacterial peritonitis	Extra-peritoneal involvement	Radiological evidence	WBC/ mm^3	Poly%	Lym	Mono	Eus
1	+	−	−	−	3200		Predominantly		Lym
2	+	−	−	−	1800		Predominantly		Lym
3	+	−	−	+	4100		Predominantly		Lym
4	+	+	−	−	500	80	−		
5	+	−	−	−	1100	18	82		
6	+	−	−	−	5000	90	−		
7	+	−	−	−	−	70	30		
8	+	−	Pulmonary liver tb	+	2200	80	20		
9	+	−	−	−	400	84	16		
10	+	−	−	−	300	−			

Poly: Polymorphs. Lym: Lymphocytes. Mono: Monocytes. Eos: Eosinophils.

Three patients died of tuberculous related complications after antituberculous treatment was started (mainly severe septic shock). One patient died of pulmonary embolism, while he was on antituberculous medications. Seven patients required catheter removal and conversion to hemodialysis. Two patients were successfully treated without the removal of Tenckhoff catheter. After admittance to the hospital because of peritonitis findings, those two patients (patients no. 5 and 7) were empirically started on vancomycin and amikacin intraperitoneally. After three weeks, antituberculous therapy was started due to repeat peritoneal fluid cell count revealed total cell counts of 300 mm3 and 450 mm3 in those two patients respectively. They had rapid clinical improvement of peritonitis after antituberculous treatment.

Prior to antituberculous therapy all patients received conventional peritonitis therapy by intraperitoneal route. The common regime was vancomycin and aminoglycoside. Eight out of 10 patients had serum albumin level of less than 35 g/L. Nutritional status and treatment of the patients before diagnosis of TBP are shown in Table 4.

Discussion

Patients with chronic renal failure have impaired cellular immunity,[27] and therefore they are susceptible to tuberculous infection. Uremic patients have a ten fold increased risk of tuberculosis infection compared to the general population.[28]

Table 3. The diagnostic methods, duration to diagnosis, treatment, and short-term outcome of the patients.

		Apparent onset to diagnosis		Short-term outcome catheter		
Patient no.	Diagnostic method(s)	(Months)	Treatment	Removal	Dialysis	Mortality
1	Smear	1	I, R, M	+	HD	Alive
2	Smear	4	I, R, M	+	HD	R
3	Smear	8	I, R, M	+	HD	Alive
4	Smear-culture	1.5	I, R, M, E	−	CAPD	NR
5	Smear-culture	1.5	I, R, P, E	−	CAPD	Alive
6	Culture	1.5	I, R, M, E	+	HD	R
7	Culture	−	I, R, M, C	−	CAPD	Alive
8	Smear+culture+ biopsy	2	I, R, E, C	+	HD	R
9	Smear	5	I, R, E, P	+	HD	Alive
10	Culture + biopsy	−	I, R, P	+	HD	Alive

Is, Isoniazid; *P*, Pyrazinamide; HD, Hemodialysis.
R, Rifampicin; *E*, Ethambutol; CAPD, Continuous Ambulatory Peritoneal Dialysis.
M, Morphazinamide; *C*, Ciprofloxacin; *R*, related TBP; NR, Non related TBP.

Table 4. Peritonitis attacks, medical treatment, and serum albumin levels of the patients.

Patient no.	Peritonitis attacks	Medical treatment before TBP diagnosis	T.protein/ albumin (g/L)
1	–	V, A, M	64/31
2	1	V, A, M, F	88/38
3	6	V, A, M	60/33
4	–	V, G	57/26
5	–	C, A, V	83/49
6	–	C, A	53/19
7	–	V, A	?
8	–	?	59/25
9	–	V, A, T	58/27
10	3	?	?

V, Vancomycin; C, Cefazolin; A, Amikacin; F, Fluconazole; M, Cefepime; G, Gentamicin.

Tuberculosis peritonitis is a rare complication. In this present study, we report 10 TBP cases with CAPD.

Tuberculous bacilli may enter the peritoneal cavity by one of the following routes: blood stream, bowel or tuberculous salpinx. In general, the pathogenesis of infection is due to reactivation of a latent focus rather than a primary infection.[29] Usually, pulmonary tuberculosis is not associated with TBP but it happens in typically young and female nonuremic patients. The average age of nonuremic with TBP patient is about 31 years.[23] Our patients were of mean age 37.2 years and TBP were seen equally in both females and males in our group.

The diagnosis of TBP is difficult because of its similarity to bacterial peritonitis. Physical examination, PDF WBC, absence of active tuberculosis, and nonspecific laboratory findings may be considered as bacterial peritonitis. Clinical presentation of TBP complicating CAPD usually includes abdominal pain, fever and cloudy effluent, which are similar to peritonitis caused by gram-positive and gram-negative bacteria and fungi. The WBC counts of PDF are not helpful either. Although in non-uremic patients with TBP, predominance of lymphocytosis in the PDF has been the usual finding in the peritoneal fluid, in TBP patients with CAPD a predominance of PNL is common.[29] TBP must be suspected in a prolonged course of ''sterile'' peritonitis unresponsive to conventional antibiotic treatment. Classically, the onset of symptoms of TBP is insidious. However, in some cases, acute symptoms may occur.[29] In our patients WBC count was unhelpful for diagnosis of TBP. Only three patients had lymphocytosis in the peritoneal fluid. The majority of our cases (68%) showed predominantly neutrophil. In the literature 78 percent of 52 cases reviewed had PNL-predominance on examination of the peritoneal fluid.[23] Concomitant/antecedent bacterial peritonitis is not an unusual finding in the patients

with TBP. Of 56 CAPD patients with TBP reported previously, 15 (27%) described concomitant/antecedent bacterial or candida peritonitis.[23,24] TBP should be considered in all cases with resistant bacterial peritonitis in which symptoms persist in spite of conventional antibacterial therapy.

Cloudy fluid and abdominal pain was invariably the presenting feature of TBP in our patients. Fever was observed in nearly half of our cases. Coincidental bacterial peritonitis was present in one patient (patient no. 4). The findings are similar to those of the cases reported so far.[6–24]

The diagnosis of TBP relies on culture. Smears of peritoneal-fluids for AFB often fail to reveal bacilli. However, a culture for AFB usually takes 6 weeks and the diagnosis of infection is delayed in most of the cases. For this reason, in the present study, the diagnosis was based on positive smear for AFB of PDF and then treatment of tuberculosis had begun. In our study, the diagnosis was made by smear in 4 and by culture in 6 patients. In 2 patients (cases no. 8 and 10) the diagnosis was established on peritoneal-fluid culture for AFB, by peritoneal biopsy. Limited laparotomy, laparoscopy, and peritoneal biopsy provide a histologic diagnosis and have been suggested as a method for making rapid diagnosis, especially in patients with sterile peritonitis unresponsive to standard antimicrobial treatment. The mean interval between onset and diagnosis of TBP was 2.3 months (range of 1–8 months) in our patients; one of the patients was in exceptional since his TBP was positive after 8 month.

A combination treatment with 3 ATDs for at least 9–12 months is necessary for successful outcome. There are few data for the optimal choice and duration of therapy of TBP. Based on the usual approach to extrapulmonary tuberculous, three antituberculous drugs recommend (rifampicin 600 mg orally, isoniazid 300 mg orally, and pyrazinamide 1.5 g orally).[30] Isoniazid and rifampicin were used in all our cases. Antituberculous drugs were given in unmodified dozen, safely and with no side effects. There is no specific recommendation on chemotherapy of tuberculosis in patients on CAPD.

Seven patients were transferred to HD after the diagnosis of TBP since TBP was resistant to effective treatment (Table 3). Two patients were successfully treated while they were on CAPD therapy. One of the patients died on CAPD because of pulmonary embolism while he was receiving antituberculous treatment. It has been known that catheter removal is necessary in TBP in patient with CAPD. We did not remove the catheter in two patients (cases no. 5 and 7) because of rapid clinical improvement of peritonitis after antituberculous treatment.

In conclusion, TBP in CAPD patients is a very rare complication, but when it occurs the morbidity and mortality rates increase dramatically. Therefore early diagnosis by rapid methods (e.g., limited laparotomy, peritoneal biopsy) and effective treatment with full dosage of ATDs are extremely important for improving outcome. In contrast to tuberculous peritonitis in nonuremic patients, CAPD patients with tuberculous peritonitis may have predominance of neutrophil on examination of the peritoneal. Also, differential leucocyte counts in peritoneal fluid may have limited diagnostic value in the differential diagnosis between tuberculous peritonitis and bacterial peritonitis.

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