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CORRESPONDENCE

A Novel Agent of Peritoneal Dialysis-Related Peritonitis: *Granulicatella Adiacens*

Editor:

Peritonitis is an important cause of morbidity and mortality in patients receiving continuous ambulatory peritoneal dialysis (CAPD). It can lead to hospitalization, switching the treatment to hemodialysis, and death. Detection of the etiologic agent(s) of the peritonitis is an important diagnostic step. In CAPD patients' peritonitis, generally 75% of episodes are caused by gram-positive micro-organisms, and *Staphylococcus epidermidis* accounts for 50% (1). Culture-negative peritonitis is a major complication for patients on CAPD and precludes organism-specific therapy. Culture-negative peritonitis has been reported in 10% – 50% of patients with CAPD-associated peritonitis (2,3). Some of the micro-organisms can grow a single colony on the culture plaque and these plaques can be reported as "no growth" because they could not be identified. This can lead a false increase in "culture-negative peritonitis" rates. Therefore, further identification must be performed. In this letter we report a case of peritonitis in a CAPD patient that was culture negative by standard techniques, but *Granulicatella adiacens* was identified when further culture systems were used.

A 55-year-old female patient was diagnosed with chronic renal failure. After 8 months of CAPD, during which she experienced two episodes of culture-positive peritonitis, she arrived at the hospital with abdominal pain and fever. In physical examination, body temperature was 38°C and there was general tenderness on her abdomen. Other physical findings were normal. She was using 2 L of peritoneal fluid 4 times per day and her dialysate was cloudy. Her laboratory findings were as follows: serum BUN 19.6 mmol/L, creatinine 521.6 μmol/L, Na 132 mmol/L, K 4 mmol/L, Ca 2.4 mmol/L, C-reactive protein 28.4 mg/dL. In her complete blood count, white blood cell count was 11300 (with 71% neutrophils, 20% lymphocytes) and hemoglobin was 12.2 g/dL. These findings made us think of peritonitis. In the dialysate, 270 leukocytes/mm^3 (100% polymorphonucleocytes) were counted and standard dialysate culture technique was performed. Thereafter we started intraperitoneal cefazolin and gentamicin empirically. Afterwards, peritoneal fluid culture was found to be negative. Peritoneal fluid was examined again with the same technique. There were 220 leukocytes/mm^3 and only one very small colony had grown on the culture plaque, but it could not be identified. For this reason further techniques were performed: *Granulicatella adiacens* was identified using the "VITEK 2 compact system" (bioMerieux, Marcy-l'Étoile, France). The organism was sensitive to cefuroxime and gentamicin. The patient received intraperitoneal cefazolin and gentamicin for 14 days. Clinical and laboratory improvement was observed in 2 days after the treatment began.

Granulicatella species are nutritionally variant streptococci, first described in 1961 by Frenkel and Hirsch (4). These gram-positive cocci were classified on the basis of growth characteristics such as nutrient requirements (pyridoxal) and presence of satellitism.

Granulicatella species form a part of the normal flora of the oral cavity, the genitourinary tract, and the intestinal tract (5–7). However, these micro-organisms cause bacteremia or local infections such as endocarditis, central nervous system infections, arthritis, and osteomyelitis (8–10). *Granulicatella adiacens* (formerly *Abiotrophia adiacens*) is isolated more frequently from oral specimens than other nutritionally variant streptococci (6). There are no reports on cases of peritonitis due to *G. adiacens*; however, we think that this agent might be responsible for peritonitis cases that were reported "culture negative" because identification was unsuccessful. Therefore, *G. adiacens* should be considered when the pathogen grows as a single small colony and cannot be identified. Identification should be performed by using such methods as the VITEK system. This system is sensitive and specific for *G. adiacens*. It is easy to use, provides early results, can provide antibiotic sensitivity tests, and is not expensive.

Diagnosis and treatment of this micro-organism is difficult because it cannot be detected by standard culture techniques and is resistant to some antibiotics (11). Our patient was fortunate because the micro-organism was not resistant to the empirically given antibiotics and treatment success was seen in a short time. The treatment was discontinued at day 14 and relapse was not

seen on the days following. It must be noted, however, that this micro-organism is resistant to some antibiotics, such as amikacin, gentamicin, penicillin, and ceftriaxone (11,12), and can cause recurrent infections. Therefore, antibiotic susceptibility patterns must be considered, the clinical findings must be closely followed, and treatment time must be planned according to clinical features. The patient must be controlled for a short time after discharge.

In conclusion, *G. adiacens* should be considered in cases of peritonitis on CAPD when the infectious agent cannot be identified and it should not be forgotten that its treatment can be difficult.

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CAPD Peritonitis Caused by *Mycobacterium Rhodesiae*

Editor:

Peritonitis is a common complication of peritoneal dialysis (PD) and a significant cause of discontinuation of PD. Peritonitis caused by nontuberculous mycobacteria (NTM) is uncommon and creates diagnostic and treatment dilemmas. We present a case of continuous ambulatory peritoneal dialysis (CAPD)-associated peritonitis due to *Mycobacterium rhodesiae*, a rapidly growing mycobacterium not previously reported to cause peritonitis.

A 50-year-old female was on PD for end-stage renal failure secondary to type II diabetes. She had been on PD for 4 years and had experienced recurrent peritonitis secondary to *Streptococcus salivarius*. All episodes were successfully treated with standard antibiotic therapy without the need for catheter removal.

The patient presented with abdominal pain and a cloudy effluent and was commenced on protocol intraperitoneal gentamicin and vancomycin. Her dialysate fluid contained 389 white cells/high power field (hpf), with 75% polymorphonucleocytes (PMNs); Gram stain was negative. Her symptoms failed to resolve and the dialysate white cell count increased. She was admitted to hospital 6 days after the onset of peritonitis. On presentation to hospital, she had symptoms of severe abdominal pain, cloudy dialysate, fever, and chills. Dialysate white cell count was 1960/hpf, with 94% PMNs. She was started on empirical fluconazole; gentamicin and vancomycin were continued. The following day the dialysate white cell count was 4640/hpf. Her Tenckhoff catheter was removed and hemodialysis was commenced.

Her symptoms improved and she was discharged the following day.

The initial dialysate was inoculated onto 5% sheep blood agar (SBA) [Fort Richard Laboratories (FRL), Auckland, New Zealand] and GC saponin agar (FRL), and into an aerobic and an anaerobic bottle from the BACTEC 9240 blood culture system (Becton Dickinson, Sparks, Maryland, USA). The cultures were incubated aerobically at 35°C. After 7 days the aerobic blood culture bottle flagged positive. No organisms were seen in the Gram stain and the culture was subcultured onto 5% SBA, Sabouraud's dextrose agar (FRL), and a glucose broth. Two days later, fine growth was seen on the SBA plate and Gram stain revealed pleomorphic gram-positive bacilli.

Due to the difficulties with culturing the isolate, 16S rDNA sequencing was carried out and the result showed a 100% match with *Mycobacterium rhodesiae*. Susceptibility testing was carried out by E-test (AB Biodisk, Solna, Sweden) using Mueller Hinton SBA (FRL) and incubated at 30°C for 5 days. The isolate was susceptible to ciprofloxacin, clarithromycin, and doxycycline, with minimum inhibitory concentrations of 0.06 mg/L, 0.12 mg/L, and 0.12 mg/L, respectively. It was resistant to sulfamethoxazole/trimethoprim.

The patient was commenced on a course of ciprofloxacin and clarithromycin that continued for 3 months. There have been no further problems with abdominal pain and she will continue on hemodialysis permanently as per patient choice.

Fungi, *Mycobacterium tuberculosis* (1), and NTM are increasingly being identified as causes of PD peritonitis. The focus of this discussion will be NTM as a cause of peritonitis in the PD setting.

The first two cases of NTM peritonitis in PD patients were reported in 1983 and both were found to have disseminated disease and were fatal. Since that time there have been several case reports of NTM peritonitis in the CAPD population (2–5); however, there have been no previously reported cases occurring due to *M. rhodesiae*. The most commonly reported cause of NTM peritonitis is *M. fortuitum* (2,5,6), a rapidly growing mycobacterium. Rapidly growing mycobacteria are those that, by definition, form mature colonies on solid agar in 7 days from subculture.

Although there are over 30 species of rapidly growing mycobacteria, 3 species, *M. fortuitum*, *M. chelonae*, and *M. abscessus* are responsible for the majority of infections in humans. *Mycobacterium rhodesiae* is a rapidly growing scotochromogenic mycobacterium that was first described in 1971. It was isolated from the sputum of patients in Zimbabwe (formally known as Rhodesia) who were suspected of having pulmonary tuberculosis (7).

Nontuberculous mycobacterial CAPD-associated peritonitis has a range of clinical presentations and is not clinically distinguishable from bacterial peritonitis. Fever, abdominal pain, and cloudy dialysate are the most common presenting symptoms and the onset may often be insidious (4). The peripheral white cell count is usually normal and the dialysate fluid white cell count is elevated, usually with a predominance of PMNs (4).

The route of infection in NTM peritonitis is likely to be via contamination of the catheter or exit site (8). It has been postulated that NTM peritonitis is more likely to occur in patients that are chronically underdialyzed and have poor residual renal function or recurrent peritonitis with repeated use of broad-spectrum antibiotics (9). The patient presented here had received several courses of antibiotics for recurrent episodes of peritonitis but not in the year preceding this episode. She was adequately dialyzed, with a weekly Kt/V of 2.28, but had no residual renal function.

Diagnosis of NTM peritonitis may be difficult and a clue may be persistently negative dialysate cultures. In most cases, the diagnosis is made by culture of the organism rather than by acid-fast smear (4,10). Peritoneal biopsy specimens may be useful and may show nonspecific chronic inflammation or granuloma formation (4,6,11).

Identification of rapidly growing mycobacteria to species levels is important as there are predictable antimicrobial susceptibility patterns. Where this is not possible, susceptibility testing using a standardized method is recommended to guide the choice of antimicrobial agent (as was carried out in this case).

Empiric therapy for NTM peritonitis with amikacin alone or in combination with cefoxitin has been suggested (4). However, newer agents, such as clarithromycin, 8-methoxy fluoroquinolones (moxifloxacin and gatifloxacin), and linezolid, have excellent *in vitro* activity against rapidly growing mycobacteria and offer an oral option for treatment (12).

Most experts suggest at least 6 weeks of treatment (4). Treatment frequently requires removal of the dialysis catheter due to failure to respond to antibiotic therapy (3,4). In one report of 15 cases, treatment was successful only after the catheter had been removed (13), but successful treatment without catheter removal has been described (14). Reported complications of NTM include intra-abdominal abscess formation, fistula formation, wound dehiscence, and intra-abdominal adhesions (10). Long-term outcomes are difficult to determine from the literature.

In summary, this is the first reported case of CAPD-associated peritonitis caused by *Mycobacterium rhodesiae* (7). Treatment consisted of catheter removal and anti-

biotic therapy, with good clinical results. Nontuberculous mycobacterium is a rare but important and treatable cause of CAPD peritonitis and should be considered in patients that are culture-negative or that fail to respond to antibiotic therapy. Optimal treatment regimens are not yet consistently defined; however, treatment should become more clear-cut as our experience increases.

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Polymicrobial Peritonitis in a Patient with Mixed Cryoglobulinemia

Editor:

Mixed cryoglobulinemia, whether associated with hepatitis C virus (HCV) infection or not, frequently produces renal involvement through leukocytoclastic vasculitis or membranoproliferative glomerulonephritis. Renal involvement is often severe, requiring renal replacement therapy. Because severe vascular and cutaneous involvement is often present, obtaining vascular access for hemodialysis can be difficult. In these patients, peritoneal dialysis is a good therapeutic alternative (1).

We report the case of a 34-year-old man, an active smoker with HCV infection without associated liver disease, who showed intolerance to interferon therapy. The patient had mixed cryoglobulinemia with multiple episodes of ischemic injuries in the lower extremities, which were treated with intravenous prostaglandin, as well as mononeuritis multiplex, which was treated with gabapentin. The patient also had end-stage renal disease secondary to membranoproliferative glomerulonephritis and recurrent urinary tract infections since childhood due to vesicoureteral reflux. Dialysis was started in September 2003 but, because of serious difficulties in obtaining vascular access, continuous ambulatory peritoneal dialysis was initiated in July 2004. The patient developed a first episode of peritonitis in October 2004 due to *Pseudomonas sp*.

The patient presented to our hospital due to a 24-hour fever with frank chills and diffuse abdominal pain. Physical examination revealed a temperature of 39°C with hemodynamic stability. The abdomen showed signs of peritonitis and the left foot showed ischemic injuries without signs of infection. The remaining physical examination was normal. Laboratory investigations revealed a septic hemogram, with 25 000 leukocytes/mL, and a C-reactive protein level of 20 mg/dL. Peritoneal fluid analysis revealed 7040 cells, with 92% neutrophils. The cryocrit was 5%, the complement levels C3, C4, and CH50 were, respectively, 117 mg/dL (normal range: 160 – 220 mg/dL), 21 mg/dL (normal: >22), and 124 mg/dL

(normal range: 160 – 220 mg/dL). Rheumatoid factor was negative. Cultures of peritoneal fluid, blood, peritoneal catheter swab, nasal swab, and ischemic injury of the left foot were performed.

A presumptive diagnosis of acute peritonitis was made and empirical intraperitoneal antibiotic therapy with vancomycin, tobramycin, and ampicillin was started before the results of peritoneal fluid culture were available. The results were obtained after 72 hours and were positive for *Enterococcus faecalis* and *Streptococcus pyogenes*. The results of the remaining cultures were negative.

The patient's initial response was favorable, with disappearance of the fever after 48 hours of antibiotic treatment. However, on physical examination, signs of peritonitis persisted. An ultrasound scan was performed, which ruled out the presence of abdominal collections. The subsequent clinical course was favorable, with progressive disappearance of clinical and laboratory signs of peritonitis.

However, because our patient had polymicrobial peritonitis due to common germs of the intestinal bacterial flora, we suspected the existence of a possible intestinal bacterial translocation. Consequently, abdominal computed tomography was performed, demonstrating wall edema of extensive segments of the small bowel, ascending colon, and part of the sigmoid colon, accompanied by vascular engorgement. These finding were compatible with intestinal injuries due to vasculitis of the small blood vessels, favoring bacterial translocation (2,3). The patient did not accept sigmoidoscopy examination.

In view of the above, we believe that the peritonitis was due to bacterial translocation in a patient with intestinal vasculitis in the context of mixed cryoglobulinemia (4,5). Importantly, despite intestinal vasculitic involvement, the patient has remained in a peritoneal dialysis program and has shown no new episodes of peritonitis or other complications, with correct dialysis parameters.

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Trichosporon asahii Following Polymicrobial Infection in Peritoneal Dialysis-Associated Peritonitis

Editor:

Fungal peritonitis is associated with decreased survival in patients receiving peritoneal dialysis (PD) (1). The majority of fungal infections are related to incorrect PD techniques (2). Among those fungi causing infections are *Trichosporon* species, which are soil and water inhabitants and cause superficial and deep infections in humans. A well-known manifestation of human Trichosporon infection is white piedra, characterized by nodules of fungal elements involving the hair shaft. *Trichosporon asahii* has been reported as the pathogen in an infection of a dialysis polytetrafluoroethylene (PTFE) arteriovenous graft (3). Localized catheter associated, systemic, or disseminated infections in immunocompromised patients have been reportedly caused by *T. asahii*, but also by *T. inkin* and *T. mucoides* (4,5). We report here the first case of continuous ambulatory peritoneal dialysis (CAPD)-related peritonitis caused by *T. asahii*.

A 49-year-old female had herb-related chronic interstitial nephritis in end-stage renal disease. She had been on CAPD since February 2003. An episode of peritonitis caused by polymicrobial infection of *Enterococcus sp* and *Streptococcus viridans* developed in December 2006. Vancomycin and gentamicin were administered intraperitoneally and the patient was then discharged in stable clinical condition.

However, the patient presented with abdominal fullness and turbid PD fluid on 10 January 2007. The abdo-

men was soft and there was no tenderness or rebounding pain. Hemogram showed white blood cell count of 9200/mm^3 with 81.7% neutrophils. Blood urea nitrogen was 51 mg/dL (18.2 mmol/L), creatinine 10.5 mg/dL (928 µmol/L), and C-reactive protein 1.07 mg/dL (normal: <0.5 mg/dL). The fluid in the peritoneal effluent bag was yellowish and cloudy, with a leukocyte count of 1350/mm^3 and 80% neutrophils. No organisms were found by Gram stain. Intraperitoneal antibiotic therapy with a loading dose of vancomycin 1500 mg was infused empirically. On day 5 after admission, a single dose of intraperitoneal fluconazole was given according to the peritoneal effluent culture, which revealed the growth of yeast. On day 6, the PD catheter was removed and a central venous catheter inserted; the patient was transferred to hemodialysis.

No significant organisms were detected in the two blood samples. The two samples of peritoneal effluent taken on the fifth day after admission revealed growth of *T. asahii*, which was sensitive to fluconazole. After treatment with fluconazole, the fever subsided and abdominal fullness improved on day 8. Fluconazole was administered intravenously for a total of 28 days during this hospital course; the patient remained in stable condition during a follow-up of 6 months.

Peritonitis is one of the most common and serious complications of PD, and most episodes are caused by staphylococci (6). Most episodes of fungal peritonitis are caused by *Candida* species, especially *C. albicans* and *C. parapsilosis*. *Trichosporon* species are causative microorganisms of superficial skin infections and they may cause either severe localized catheter-related or systemically disseminated infections in immunocompromised patients (4,5). The first reference to Trichosporon peritonitis in a patient undergoing CAPD was reported by Khanna *et al.* (7). The first case of *Trichosporon inkin* peritonitis treated with caspofungin was reported by Madariaga *et al.* (8). The other five species are *T. ovoides*, *T. cutaneum*, *T. asteroides*, *T. asahii*, and *T. mucoides* (5). *Trichosporon asahii* is a pathogen in patients with immunodeficiency (4,9) and its infection of an arteriovenous graft was reported in a hemodialysis patient, who was successfully treated with fluconazole and surgical removal of the graft (3).

There is a paucity of data on the susceptibility of *T. asahii* to antifungal agents. As azoles (including miconazole, itraconazole, ketoconazole, and voriconazole) have higher *in vitro* activity than amphotericin B, they should be considered as the antifungal drug of choice for the treatment of Trichosporon peritonitis.

In conclusion, a combination of early initiation of antifungal therapy and removal of the peritoneal catheter may lead to a better prognosis. Although rare, *Trichosporon* species still need to be considered in the diagnosis of fungal peritonitis in CAPD patients, especially in those patients with a new episode of peritonitis after remission of a previous episode of polymicrobial peritonitis treated by combination therapy of broad-spectrum antibiotics.

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