Mycobacterium fortuitum Peritonitis Associated with Continuous Ambulatory Peritoneal Dialysis

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Received 12 November 1985/Accepted 2 January 1986

Mycobacterium fortuitum has been isolated from skin and soft tissue lesions with increasing frequency. Rarely, however, has it been documented as a cause of peritonitis in patients receiving continuous ambulatory peritoneal dialysis. We report here the second such case and discuss both the possibility of M. fortuitum or similar organisms as one cause of “sterile” peritonitis in this patient population and the in vitro antimicrobial susceptibility testing of such isolates.

Mycobacterium fortuitum has been isolated as a pathogen from several extrapulmonary sites. There has been, however, only one well-documented case of M. fortuitum causing peritonitis in a patient receiving continuous ambulatory peritoneal dialysis (CAPD) (13). We present here the second such case. The possible role of the organism in culture-negative peritonitis and methods of determining the in vitro antimicrobial susceptibilities of such isolates are discussed.

Case report. A 32-year-old male being treated with CAPD for end-stage renal disease and with several medications for various psychiatric problems was admitted on 18 February 1985 with complaints of confusion and visual hallucinations. CAPD had been initiated in July 1984, and since that time the patient had experienced at least four episodes of peritonitis. On two occasions no organisms were cultured; Staphylococcus epidermidis and S. aureus were each cultured during two separate episodes.

A physical examination at admission revealed a disoriented, thin male. His blood pressure was 134/90, his pulse was 88 beats per min and regular, and his temperature was 37.5°C. His abdomen was soft, and the catheter site was clean. The peripheral leukocyte count was 5,000/mm³, with a normal differential. The patient improved after psychotropic drugs were stopped, but he remained hospitalized to receive intensive psychotherapy. CAPD was continued.

On 28 February 1985, the patient’s temperature rose to 38.3°C, and he complained of abdominal pain. The peritoneal fluid was cloudy and contained 3,845 leukocytes per mm³, with 83% polymorphonuclear leukocytes. No organisms were revealed by Gram staining. The peripheral leukocyte count was 8,400/mm³, with a slight left shift. Vancomycin and rifampin were administered after routine culturing of the peritoneal fluid was done. These cultures yielded no organisms. Fever and abdominal pain persisted, and the peritoneal fluid remained cloudy. The fluid was submitted for routine culturing as well as for culturing for fungi and mycobacteria on 3 March and 5 March. Routine cultures again showed no growth, and Gram staining revealed no organisms. Rifampin was discontinued, and gentamicin was begun. On 8 March, the peritoneal catheter was surgically removed, samples for culturing were taken of peritoneal fluid, peritoneal tissue, and the exit site, and a peritoneal biopsy was obtained. Histologic examination of the latter showed nonspecific chronic inflammation. On 9 March, the fluid submitted 6 days before was reported to be “positive for acid-fast bacilli, probably not Mycobacterium tuberculosis.” Isoniazid, ethambutol, and rifampin were begun. Amikacin was added when the exit-site sample and fluid taken at surgery grew rare numbers of colonies of Pseudomonas aeruginosa. The patient was discharged on these medications on 16 March.

On 22 March, the acid-fast bacillus was identified as M. fortuitum. Disk diffusion susceptibility studies showed the organism to be resistant to gentamicin, tobramycin, erythromycin, and sulfamethoxazole-trimethoprim, as no zone was present around the disks for these drugs. The zone diameters measured 12 mm for tetracycline and 20 mm for amikacin. The same organism was also cultured from fluid submitted on 5 March, from peritoneal tissue, and from the exit-site sample. Antituberculous drugs were discontinued, and therapy was changed to tetracycline and amikacin. The patient refused to take the latter after approximately 1 month because of decreased audio acuity. In May 1985, a fistula draining clear fluid developed above the abdominal wound. A culture of the fluid grew M. fortuitum. Ciprofloxacin was begun after susceptibility studies indicated in vitro activity. Over the next several months, drainage from the fistula decreased; however, a sample taken on 12 September remained culture positive for M. fortuitum. Repeat susceptibility studies showed an identical pattern.

Results. In our laboratory, peritoneal fluid submitted for culturing from CAPD patients is received in 2-liter quantities. From this volume, 100 ml is removed aseptically and centrifuged for 15 min at 3,500 rpm. The sediment is routinely planted on sheep blood agar, chocolate agar, MacConkey agar, and thioglycolate broth and incubated at 35°C for 48 h. A cytopsin preparation for Gram staining is also made. For acid-fast cultures, the sediment is planted on Lowenstein-Jensen, Mycobactosel (BBL Microbiology Systems), Middlebrook 7H11, and Mitcheson agars, incubated at 35°C in CO₂ and observed for 8 weeks.

M. fortuitum was isolated in large numbers from this patient’s peritoneal fluid on several occasions as well as from peritoneal tissue, the peritoneal catheter exit site, and an abdominal fistula tract. Growth was apparent in 6 to 10 days. Individual colonies were smooth and buff colored in the dark and after exposure to light. The organism grew equally well at 26, 37, and 42°C. The following biochemical reactions were observed: negative for niacin; positive for nitrate, urea, Tween 80 hydrolysis, catalase at 68°C, tellurite reduction, growth on MacConkey agar without crystal violet, 3-day arsulfatase, iron uptake, and growth in 5% NaCl.

Disk diffusion susceptibility testing was performed (19).
Several colonies of the isolate were emulsified in Middlebrook 7H9 broth with beads and incubated at 37°C in 5 to 10% CO₂ until the turbidity exceeded that of a McFarland 0.5 barium sulfate standard (approximately 3 days). The turbidity was adjusted to match that of a McFarland 0.5 standard, and a 10⁻² dilution of the culture was made in 7H9 broth. The quality control organisms M. fortuitum (Trudeau collection 1529) and M. chelonae (Trudeau collection 1542) were treated in an identical manner. Separate swabs from the 10⁻² dilution were used to streak the surfaces of each of three agar plates: Mueller-Hinton, Mueller-Hinton with 5% sheep blood, and Middlebrook 7H11. The antimicrobial disks placed on each plate included tetracycline (30 µg), tobramycin (10 µg), gentamicin (10 µg), amikacin (30 µg), sulfamethoxazole-trimethoprim (23.75 µg:1.25 µg), and ciprofloxacin (5 µg). The plates were incubated at 37°C in CO₂ and read at 48 and 72 h. On Mueller-Hinton agar, there was no zone of inhibition for gentamicin, tobramycin, erythromycin, or sulfamethoxazole-trimethoprim. The zone diameters were 12 mm for tetracycline, 30 mm for amikacin, and 50 mm for ciprofloxacin. The zone diameters differed slightly on the other media, but with one exception this did not alter the final interpretation. On Mueller-Hinton agar supplemented with blood, the zone diameter for sulfamethoxazole-trimethoprim was 30 mm at 48 h, but at 72 h no zone was present.

Discussion. Peritonitis continues to be a serious problem in patients receiving CAPD and is the major cause for transfer to other methods of dialysis (12). In our experience, coagulase-negative staphylococci are the most frequent isolates, followed by S. aureus. Various gram-negative bacilli account for 15 to 20% of the isolates, and yeasts account for approximately 5% (unpublished data). These results are similar to those of others (4, 5, 7, 14, 15, 18). In addition, anaerobes are rarely isolated.

The incidence of “sterile” peritonitis varies from 4 to 37% (4, 7). There are at least three reasons why this might occur. First, true “aseptic” peritonitis does seem to exist. These patients have high numbers of eosinophils in their peritoneal fluid and respond to steroid therapy (6, 10). Second, the sensitivity of the method used to culture the peritoneal fluid may influence organism recovery. Techniques include centrifugation of a certain volume of fluid followed by culturing of 0.5 ml of the supernatant on a specific medium (e.g., Columbia agar) or filtration of a specific volume of fluid through a filter (15, 18), and inoculation of a portion of the fluid directly into blood culture bottles (7). Recently, Dawson et al. described a technique that involved culturing of the total volume of fluid, which in their experience had a sensitivity of 100% (4). A third reason for culture-negative peritonitis is the nature of the causative organism. Various fungi and mycobacteria might be missed when specimens are processed as previously described. Since patients with end-stage renal disease do have an increased susceptibility to infections with mycobacteria (1, 16), it is not surprising that these organisms would account for a percentage of cases of sterile peritonitis in CAPD patients. Several cases caused by M. tuberculosis have been described (9, 11); however, to our knowledge, there has been only one other well-documented report of M. fortuitum peritonitis in a CAPD patient (13). M. fortuitum has, however, been mentioned as a cause of infection in association with a peritoneal catheter (20). An outbreak of peritonitis caused by an M. chelonae-like organism which was cultured from peritoneal dialysis machines used for chronic intermittent dialysis has also been described (2).

M. fortuitum and M. chelonae are environmental organ-isms that have been increasingly recognized as potential pathogens within the past several years. Although both can cause pulmonary and disseminated illnesses, they are now being isolated more frequently from primary skin and soft tissue infections, from postsurgical infections, especially those associated with augmentation mammoplasty and median sternotomy, and from abscesses after injections or other types of trauma (21). The mainstay of therapy is surgical debridement, if possible, but without chemotherapy the infection may persist for months to years. In general, these organisms are resistant to antituberculous agents; however, several recent in vitro studies in which both disk diffusion and microtiter dilution methods were used have shown that a number of traditional antibiotic agents do have significant activity against these organisms at achievable serum levels (17, 19, 20, 22). For example, studies have shown M. fortuitum to be susceptible to sulfamethoxazole, amikacin, cefoxitin and, in approximately 50% of cases, to doxycycline. M. chelonae, however, is somewhat more resistant to these same drugs (20). Newer antimicrobial agents, such as imipenem and ceftizoxime, also appear to have good in vitro activity (3). Although standards for the disk diffusion test have not yet been formulated, various publications have suggested susceptibility zone diameters (2, 19–21). For tetracycline, this diameter is 25 mm. In light of this, the tetracycline zone size of 12 mm observed in this report would indicate that, in vitro, the organism was not susceptible.

In summary, we reported a case of peritonitis caused by M. fortuitum in a CAPD patient whose initial peritoneal fluid cultures were sterile. This serves to emphasize the fact that culturing for organisms such as mycobacteria should be done for CAPD patients with clinical evidence of peritonitis but for whom routine culturing repeatedly yields no organisms.

LITERATURE CITED