Campylobacter jejuni Peritonitis Complicating Continuous Ambulatory Peritoneal Dialysis

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We report the occurrence of Campylobacter jejuni peritonitis complicating C. jejuni enteritis in a patient treated with continuous ambulatory peritoneal dialysis. Cure followed oral administration of erythromycin and intraperitoneal therapy with gentamicin.

Since the introduction of continuous ambulatory peritoneal dialysis (CAPD) in end-stage renal failure, peritonitis remains the main complication of this form of therapy. The present incidence of peritonitis in CAPD-treated patients ranges from one episode per 10 patient weeks (12) to one episode per 10.5 patient months (10).

Microorganisms involved in this particular type of peritonitis are usually of cutaneous origin (Staphylococcus epidermidis, S. aureus, Streptococcus sp.). This was true for 55% of clinical peritonitis cases reported in one study (13). Gram-negative bacteria occur less frequently (15%) and are usually observed later in the course of CAPD therapy. The remaining 30% of peritonitis cases are most often aseptic. Hence, most of the time, antibiotic administration (cephalosporin in the United States and trimethoprim/sulfamethoxazole [SMT] in Europe) is started as soon as peritonitis is suspected, before the results of bacteriological investigations are known. However, culturing of the peritoneal effluent remains imperative since unusual microorganisms may be involved (4, 6), necessitating the use of other drugs. Accurate identification of the causal pathogen may also lead to determining the site of origin of the peritonitis; for example, anaerobes would strongly suggest intestinal perforation. We report here the first case, to our knowledge, of Campylobacter jejuni peritonitis occurring in a CAPD-treated patient.

Case report. The patient, a 50-year-old man with end-stage chronic glomerulonephritis, was admitted to the hospital for hemodialysis in November 1977. In March 1978 he received a cadaver kidney transplant which was rejected after 1 month, and he was re-admitted to the hospital for hemodialysis. In April 1979 he had to be shifted to CAPD for recurrent thrombosis of several arteriovenous fistulae. Since the initiation of CAPD, four episodes of peritonitis occurred, the last one, due to Streptococcus mitior, taking place 9 months before the latest admission.

On 4 October 1981 the patient developed diarrhea with low-grade fever and abdominal discomfort. On 5 October he was admitted to the hospital because of a cloudy peritoneal effluent. Diarrhea was still present, the temperature was 37.4°C, the heart rate was 108 beats per min, and the blood pressure was 105/85. The abdomen was diffusely tender, with generalized rebound pain, but intestinal peristalsism was present. The peritoneal fluid contained 1,330 leukocytes per mm3 and a protein level of 314 mg/dl; a Gram-stained smear of the dialysate was negative. Proper portions of stools, blood, and peritoneal effluent were obtained for culturing; SMT (80 mg of sulfamethoxazole and 16 mg of trimethoprim per liter) was added to the dialysis bags, and CAPD was carried out in the usual way. No clinical improvement followed, and the peritoneal effluent remained cloudy. On day 4, the initial peritoneal fluid culture grew C. jejuni; this bacterium was also recovered from stool cultures. SMT was replaced by gentamicin (8 mg/liter) in dialysis bags, and erythromycin (3 g orally per day) was administered. Within the next 48 h, the peritoneal fluid cleared, and gastrointestinal symptoms subsided. Three sets of blood cultures obtained upon admission remained negative. Seroconversion for C. jejuni was demonstrated 2 weeks later. A barium enema examination disclosed no abnormality.

We routinely use horse blood agar plates and modified Lombard-Dowell broth (7) for culturing peritoneal fluid obtained from CAPD patients. In the present case, blood plates yielded...
no growth, but broth cultures yielded small, Gram-negative, curved rods which were further identified as C. jejuni.

For blood cultures, we proceeded as follows. Ten milliliters of blood was aseptically drawn and equally distributed in two bottles: an anaerobic bottle (brain heart infusion broth containing p-aminobenzoic acid, vitamin K, hemin, and sodium polyanetholesulfonate [50 ml; GIBCO Laboratories]) and an aerobic bottle (diphasic Castaneda medium [50 ml; Pasteur Institute, Lille]). Sets of blood cultures were incubated for 10 days at 35°C. Bottles were macroscopically examined every day, and blind subcultures were systematically performed, 12 to 24 h after the specimen was received, on chocolate agar which was incubated for 3 days in a 5% CO₂-enriched atmosphere. In the present case, a supplementary blind subculture was done on day 5 of incubation.

The antibody titer was determined by S. Lauwers (Vrij Universiteit, Brussels), who used a tube agglutination method. A formalinized antigen was prepared from strains isolated from stools and from peritoneal fluid. Similar results (titers of <1:80 upon admission and 1:2,560 2 weeks later) were obtained with both antigenic preparations.

Discussion. C. jejuni is presently recognized as a major agent of acute enteritis in humans all over the world (1). With appropriate culture techniques, it is found as frequently as salmonellae or shigellae, being recovered from 5.1 to 7.1% of diarrheic stools (2, 17). Healthy carriers are rare and are found in the vicinity of symptomatic patients (17). Transmission takes place by the fecal-oral route through contaminated food or feces (1). Septicemia rarely follows transfusion of infected blood (11).

In vitro, C. jejuni is usually susceptible to erythromycin and aminoglycosides and resistant to penicillins and cephalosporins (5 to 92% of strains produce a beta-lactamase (16, 21); susceptibility to SMT is variable (19). In our patient, who was first treated intraperitoneally with SMT, peritoneal fluid cultures remained positive until gentamicin was added to the dialysate.

The pathogenesis of CAPD peritonitis is not always obvious. In most cases, it follows a septic manipulation in connecting the bag or the tubing to the peritoneal catheter. Less frequently, it is secondary to an abscess at the entry site of the catheter. Contamination of the peritoneal cavity could also follow transmural migration of intestinal bacteria after alterations in the peritoneal wall or intestinal wall or both, owing to their permanent bathing with dialysate (15, 18). In fact, in at least one study (17), peritonitis was due in 26% of cases to intestinal bacteria. In

our patient, C. jejuni was found in both peritoneal fluid and stools, but blood cultures were negative.

Transmural migration of the microorganisms was probably responsible for peritonitis, along with the invasive capacity of C. jejuni, which has been previously described (G. M. Ruiz-Palacios, E. Escamilla, and N. Torres, Program Abstr. Intersci. Conf. Antimicrob. Agents Chemother. 20th, New Orleans, La., abstr. no. 697, 1980). It is known that C. jejuni enteritis may lead to bacteremia (3, 5), but its actual incidence remains unknown. Hence, contamination of the peritoneal cavity may have followed transient bacteremia even though blood cultures upon admission were negative. In fact, similar cases of localized infections due to C. fetus subsp. intestinalis, probably secondary to unrecognized bacteremia, have been described in the literature: arthritis (9), pericarditis (8), and, more recently, peritonitis in a CAPD-treated patient (20). Another possibility is that the patient introduced the bacteria through septic manipulation, but this is unlikely, because in this patient, only four episodes of peritonitis produced in this way occurred in the course of 30 months.

One case of C. jejuni peritonitis was reported in an 83-year-old man with heart failure, ascitic liver cirrhosis, and C. jejuni bacteremia (14). The present patient probably represents the first reported case of C. jejuni peritonitis occurring in the course of C. jejuni enteritis during CAPD treatment.

LITERATURE CITED


