Dear Sir,

Peritonitis is the major complication of continuous ambulatory peritoneal dialysis (CAPD). As devices and technology have been improved to prevent peritonitis, its prevalence in patients on CAPD has gradually declined. However, some patients with peritonitis are subject to prolonged hospitalization and catheter loss, and eventually transfer to temporary or permanent hemodialysis. Many species of microorganisms have been reported as causative agents of CAPD peritonitis. We have experienced a case of peritonitis caused by *Campylobacter fetus* subsp. *fetus* in a patient with chronic renal failure on CAPD.

A 47-year-old man with end-stage renal failure due to chronic glomerulonephritis was admitted for CAPD therapy in June 1989. In May 1990, he experienced an episode of exit site infection caused by *Staphylococcus aureus*, and was successfully treated with vancomycin. In July 1990, he complained of abdominal pain, sore throat, low-grade fever (37.7°C), diarrhea, and cloudy peritoneal effluent. He had active bowel sounds throughout, without rebound pain or guarding. The blood leukocyte count was 7,900/µm³, 59% of which were neutrophils and 12.5% juvenile forms. The peritoneal fluid contained 989 leukocytes/µm³, 90% of which were neutrophils and 5% juvenile forms. He was given 1 g of vancomycin intraperitoneally. The peritoneal fluid was obtained for cultures. Three days later, he visited our hospital again, complaining of persistent cloudy effluent, although the subjective symptoms had been ameliorated. On the 7th day after onset of peritonitis, the initial peritoneal fluid culture was found to be growing *C. fetus* subsp. *fetus*. Cultures of the peritoneal fluid from the 4th day also yielded the same organism. Intravenous injection of 2 g cefmetazole sodium and oral administration of 600 mg norfloxacin were started on the 7th day. Three days later, the leukocyte count in peritoneal fluid was decreased to 42/µm³, and peritoneal fluid culture was negative. Multiple blood and urine cultures were negative throughout the period. Intravenous cefmetazole sodium was given for 7 days and oral norfloxacin for 14 days. Peritoneal fluid cultures were negative thereafter.

Most episodes of peritonitis in CAPD patients are due to normal skin flora, i.e. *S. epidermidis* and *S. aureus*, and a smaller fraction of peritonitis episodes are caused by gram-negative organisms, presumed to originate from the bowel [1]. However, peritonitis is sometimes caused
by unusual organisms. Campylobacter rarely causes peritonitis in CAPD. As far as we know, 4 cases of peritonitis caused by C. jejuni and only 2 cases caused by C. fetus subsp. fetus have been reported [2-7]. Campylobacter, a common pathogen in the genital and intestinal tracts of sheep and cattle, was first isolated in humans in 1947 [8]. Campylobacter was identified in 1973 as two, and, more recently, three separate species and subspecies (C fetus subsp. fetus, C. jejuni, and C. coli) [9] associated with two distinct clinical syndromes. First, C. jejuni and C. coli have been recognized as common intestinal pathogens in previously healthy persons [10]. Second, C. fetus subsp. fetus is an infrequent cause of human infection, which usually produces a systemic illness (i.e., meningitis, bacteremia), and is most often isolated from blood cultures [11]. The mode of acquisition of C. fetus infection is not known in most cases. In our case, the patient had a habit of ingesting raw liver. Therefore, it is likely that the bacteria migrated to the abdominal cavity through the inflamed intestinal wall. C. fetus subsp. fetus infection occurs predominantly in patients with debilitating or immunosuppressive disease [12]. The end-stage renal disease of this patient was likely an underlying condition of the infection. Erythromycin has been frequently used in reported cases [13]. In our case, cefmetazole sodium and norfloxacin to which bacteria are susceptible in vitro were used, because failure has been reported in an erythromycin-treated case [13]. Although relapse has been reported in some cases [5,13], our patient has been well for 2 months since the cessation of antibiotic therapy.

In summary, nephrologists should be aware of the potential for pathogenicity of C. fetus subsp. fetus in CAPD patients, and of its prompt diagnosis and appropriate therapy.

References


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C. fetus. Subsp. fetus Peritonitis in CAPD