Lesser Sac Abscess Induced Relapsing Peritonitis in a CAPD Patient

Chin-Huang Chen^a Yi-Hong Chou^b Chui-Mei Tiu^b Chun-Cheng Hou^c Chang-Linct Yang^c
Yee-Yung Ng^c Wu-Chang Yang^c

^aDepartment of Internal Medicine, Tsu-Tong Veterans Hospital; ^bDepartment of Radiology, and

^cDivision of Nephrology, Department of Internal Medicine, Taipei Veterans General Hospital, and National Yang Ming University, Taipei, Taiwan, ROC

Dear Sir,

Peritonitis is a common complication in patients with end-stage renal disease treated by continuous ambulatory peritoneal dialysis (CAPD). It is usually caused by poor technique, exit-site infection [1, 2], tunnel infection [3, 4], perforation of hollow viscera [5], diverticulitis [6, 7], colitis [8], or cholecystitis [9], etc. It is usually easily treated by effective and adequate antibiotics in most cases. However, it becomes complicated and protracted in some cases if the uncommon abdominal abscesses cannot be found and eradicated completely [10]. However, repeated episodes of peritonitis attributed to isolated lesser sac abscess have never been reported. Herein, we report a rare case of isolated lesser sac abscess formation following repeated episodes of peritonitis in a CAPD patient. The clinical condition improved dramatically after ultrasound-guided aspiration of the lesser sac abscess.

A 38-year-old woman was under CAPD therapy for 57 months. She was admitted to hospital because of epigastric pain developed and progressed to diffuse abdominal pain in the morning. It was followed by turbid dialysate effluent. The white cell count (WBC) of the effluent was 480/cm³. The peritoneal effluent culture was positive for coagulase-negative staphylococcus (CNS). Three flushings of heparinized 1.5% Dianeal

dialysate were given and followed by IP antibiotics (cefazolin loading 500 mg/l, maintenance 125 mg/l and tobramycin loading 8 mg/l, maintenance 4 mg/l) immediately. The cloudy effluent became clear and WBC decreased to 1/mm³ on the 4th day. The antibiotic therapy was administered for 2 weeks. Two months later, similar symptoms and peritonitis recurred. The WBC of the effluent was 2,900/mm³. The culture of the effluent also showed CNS, which was also sensitive to cefazolin and gentamicin. The three flushings and antibiotics as above were given for 2 weeks, even though the cloudy effluent became clear and WBC of the effluent declined to 37/mm³ on the second day. However, a 3rd episode of peritonitis relapsed 1 month later. Since then, the frequency of relapsing peritonitis has increased and the interval between peritonitis episodes has been shorter even under persistent antibiotic therapy (fig. 1). The symptoms of each episode presented similarly. The pathogen of each episode of peritonitis was CNS with the same bacterial sensitivity test.

On physical examination, there was no erythema or discharge from the exit site, no evidence of acute tunnel infection, no rebound pain, and no Murphy's sign. The WBC was 6,200/mm³ with 62% neutrophils. The hematocrit was 32%. Platelet count was

283,000/mm³. Serum biochemistry showed calcium 8.9 mg/dl (reference range: 8.4–10.6), inorganic phosphate 4.0 mg/dl (2.1–4.7), AST 20 U/l (5–45), ALT 3 U/l (0–40), amylase 221 U/l (<190), lipase 178 U/l (<190). Computed tomography (CT) of the whole abdomen before drainage of dialysate showed no evidence of diverticulitis, space-occupying lesions or enlarged lymph nodes. The upper gastrointestinal (UGI) and small intestine series did not show peptic ulcer or any abnormality. The subsequent Tc99m-labeled WBC scanning for detection of intra-abdominal abscess showed a negative result. Abdominal sonography after dialysate drained out revealed fluid accumulation in the lesser sac. Because there were 4 other episodes of turbid effluents happening under the antibiotic coverage after the above examinations, a 2nd sonography of the whole abdomen after dialysate drained out was arranged which also revealed fluid accumulation, sized 3 × 2 × 1.5 cm in the lesser sac (fig. 2). At this time, ultrasound-guided aspiration of the lesser sac fluid was done with the finding of 15 cm³ of turbid aspirate. The aspirated fluid culture was positive for CNS. The bacterial sensitivity test was the same as before. Eight days later, follow-up CT of the whole abdomen after the dialysate drained out showed no evidence of focal fluid accu-

Fig. 1. Clinical course of relapsing peritonitis induced by lesser sac abscess. Arrow = Episodes of peritonitis; CT = computerized tomography of whole abdomen; Sono = sonography of whole abdomen; UGI series = upper gastrointestinal series and small intestine; arrowhead = ultrasound-guided aspiration of lesser sac fluid.

mulation in the lesser sac. The antibiotics were discontinued 2 weeks later. In the following 2 years, there was no more peritonitis.

In this case, the frequently relapsing peritonitis indicated that there was an occult infective source not related to inadequate or inappropriate therapy. The culture of aspirate fluid from the lesser sac showed CNS with the same bacterial sensitivity test as previous dialysate culture. There was no more peritonitis after the pus was aspirated from the lesser sac. So the sonogram findings and clinical course support that the infectious source of relapsing peritonitis came from the lesser sac abscess.

In this case, the 1st abdominal CT did not find any focal fluid accumulation in the lesser sac. This might be because the dialysate fluid was not drained out during the CT examination. In the study of Twardowski et al. [11], they mentioned that approximate fluid contents in the lesser sac were 1–3% (20 cm³ for a 2-liter dialysate) before drainage as appraised in the supine position. Usually, there is no more fluid accumulation in the lesser sac after drainage in both the supine and the erect positions [11]. Therefore, the repeated abdominal sonograms were performed after dialysate drainage and showed fluid collection in the lesser sac. Dialysate drained out before CT or sonogram examination was important for the diagnosis of abdominal and retroperitoneal abscesses.

The characteristic and consistent epigastric pain progressive to diffuse abdominal pain in the morning in each episode of peritonitis may be a helpful clue of lesser sac abscesses. However, it may need further cases for evaluation.

Singh and Wadhwa [12] reported a mortality CAPD case with concurrence of protracted peritonitis, pancreatitis, and infected pancreatic pseudocyst or lesser sac abscess. They addressed two proposed sequences of events: (1) pancreatitis as the initial event,

Fig. 2. Sonography demonstrated fluid space corresponding to the lesser sac (arrowheads). Internal echoes of the fluid are suggestive of infection or abscess formation. ST = Stomach.

then pseudocyst formation, infected pseudocyst, overt peritonitis, and (2) incomplete resolution of peritonitis as the initial event, then lesser sac abscess formation followed by overt peritonitis and acute pancreatitis. However, there was no evidence of pancreatitis in our case because of no significant elevation of serum amylase or lipase. Abdominal CT and sonogram also did not show any evidence of pancreatitis. This means that lesser sac abscess can cause overt peritonitis without pancreatitis.

In conclusion, when there is early-morning epigastric pain followed by turbid dialysate effluent in CAPD patients, lesser sac abscess formation should be included in the differential diagnosis of abdominal events in CAPD patients. Ultrasonography or CT scan of the abdomen should be performed after the dialysate is drained out. In addition to adequate and effective antibiotic therapy, aspiration of the lesser sac abscess should be considered for complete eradication of peritonitis.

Acknowledgements

This work was supported by grants from the National Science Council of Taiwan (NSC 89-2314-Bo75-153).

References

- 1 Piraino B, Bernadini J, Sorkin M: A five-year study of the microbiological results of exit site infection and peritonitis in continuous ambulatory peritoneal dialysis. *Am J Kidney Dis* 1987; 10:281–286.
- 2 Gucek A, Benedik M, Zakelj B, Stanisavljevic D, Lindic J, Hergouth V, Bren AF: Frequency of various types of peritoneal catheter infection and therapeutic outcome of treatment. *Adv Perit Dial* 1995;11:149–151.
- 3 Tzamaloukas AH, Hartshorne MF, Gibel LJ, Murata GH: Persistence of positive cultures after apparent cure of CAPD peritonitis. *Adv Perit Dial* 1993;9:198–201.
- 4 Holley JL, Bernardini J, Piraino B: Risk factors for tunnel infections in continuous peritoneal dialysis. *Am J Kidney Dis* 1991;18:344–348.
- 5 Wakeem MJ, Zimmerman SW, Bidwell D: Viscus perforation in peritoneal dialysis patients. *Perit Dial Int* 1994;14:371–377.
- 6 Wu G, Khanna R, Vas S, Oreopoulos DG: Is extensive diverticulosis of the colon a contraindication to CAPD? *Perit Dial Bull* 1983;3:180–183.
- 7 Steinberg SM, Cutler SJ, Nolph KD, Joel WN: A comprehensive report on the experience of patients on continuous ambulatory peritoneal dialysis for management of end stage renal disease. *Am J Kidney Dis* 1984;4:233–241.
- 8 Wehling M, Jenni R, Steurer J, Buhler H, Siegenthaler W, Kuhlmann U: Ischaemic colitis in a patient undergoing continuous ambulatory peritoneal dialysis. *Perit Dial Bull* 1982;2:123–124.
- 9 Nelson W, Khnana R, Mathews R, Yeung H, Wu G, Vas S, Oreopoulos DG: Gall bladder stones, cholecystitis and cholecystectomy in patients on continuous ambulatory peritoneal dialysis. *Perit Dial Bull* 1984;4:245–248.
- 10 Boroujerdi-Rad H, Juergensen P, Mansourian V, Kliger AS, Finkelstein FO: Abdominal abscesses complicating peritonitis in continuous ambulatory peritoneal dialysis patients. *Am J Kidney Dis* 1994;23:717–721.
- 11 Twardowski ZJ, Tully RJ, Ersoy FF, Narandra MD: Computerized tomography with and without intraperitoneal contrast for determination of intraabdominal fluid distribution and diagnosis of complications in peritoneal dialysis patients. *Trans Am Soc Artif Intern Organs* 1990;36:95–103.
- 12 Singh S, Wadhwa N: Peritonitis, pancreatitis, and infected pseudocyst in a continuous ambulatory peritoneal dialysis patient. *Am J Kidney Dis* 1987;1:84–86.

Announcement

3rd International Congress on Uremia Research

The 3rd International Congress on Uremia Research will take place in Taormina, Sicily, on October 2–4, 2003. Deadline for submission of abstracts is May 2, 2003. For further information, please contact Prof. Guido Bellinghieri, Viale Regina Margherita, 69, I-98100 Messina (Italy). Fax: 39 090 36 36 26.