PART TWO

Host Defenses and Infection
Transient bacteremia during and after endoscopic procedures is a well-documented phenomenon. *Streptococcus viridans* peritonitis is frequently associated with peritoneal dialysis, and the infection is probably attributable to hematogenous spread, dental procedures, or transluminal contamination with oral flora. To our knowledge, no reports exist of peritonitis occurring after gastroscopy in peritoneal dialysis patients.

Here, we report the case of a 69-year-old male patient receiving automated peritoneal dialysis who required emergency gastroscopy and sclerotherapy plus heat-probe coagulation to control active bleeding from a duodenal ulcer. The next day, this patient developed nausea and abdominal pain. The diagnosis of peritonitis was made based on a cloudy peritoneal effluent and a leukocyte count of 11,500 cells/μL with 98% neutrophils. *S. viridans* was identified in the peritoneal fluid culture. The patient received cefazidime for 14 days, followed by clarithromycin for 7 days, and he recovered successfully. Patients receiving peritoneal dialysis who undergo esophagogastro-duodenal endoscopy are at risk to develop peritonitis, and so antibiotic prophylaxis is desirable.

**Key words**

*Streptococcus viridans*, peritonitis, gastroscopy

**Introduction**

Transient bacteremia during and after endoscopic procedures is a well-documented phenomenon. High-risk procedures include esophageal dilation and esophageal sclerotherapy. Lower-risk interventions include gastroscopy, sigmoidoscopy, and colonoscopy. The most common infection occurring after variceal injection sclerotherapy is bacterial peritonitis, which in large series of cirrhotic patients occurred at a mean frequency of 3.7%. Other infectious complications associated with esophageal sclerotherapy and dilations have included rare cases of endocarditis, central nervous system infection, pyrogenic arthritis, and a perinephric abscess (1,2). Most organisms isolated in blood cultures are mouth commensals such as *Streptococcus viridans* (3).

A review of recent literature demonstrates that lower gastrointestinal endoscopy, gynecologic procedures, and dental procedures performed without prophylaxis in patients receiving peritoneal dialysis (PD) can lead to peritonitis. The most common causative pathogens were *S. viridans* and gram-negative rods (4). In PD-associated peritonitis, *S. viridans* is isolated at a frequency of 10% – 15%. Infections are probably caused by hematogenous spread, dental procedures, or transluminal contamination with oral flora (5,6). Here, we report what we believe to be the first case of peritonitis after gastroscopy in a PD–treated patient.

**Case report**

A 69-year-old Hispanic man with end-stage renal disease secondary to hypertensive nephrosclerosis had been undergoing automated PD (APD) for 21 months. He had had an episode of *Pseudomonas stutzeri* peritonitis two months after the initiation of APD and had been treated successfully with ceftazidime and ciprofloxacin. The infection did not recur, and there was no need for catheter removal.

Because of obstructive symptoms caused by prostatic adenoma, this patient required suprapubic radical prostatectomy. One week after discharge, he was brought to the emergency room because of syncope. He denied fever, nausea, vomiting, or abdominal pain. Physical examination showed intense pallor, blood pressure of 122/57 mmHg, pulse of 83 beats per minute, and normal body temperature. On examination the heart, lungs, and abdomen were unremarkable. No focal neurological deficits were observed. Laboratory results disclosed a hematocrit of 15%, a platelet count of 324,000/mm³, a prothrombin time of 10.2 s, and a partial thromboplastin time of 31.3 s.
Screening for occult gastrointestinal blood loss was positive. Emergency gastroscopy revealed a duodenal ulcer with active bleeding (Forrest II-B). An epinephrine injection, plus heat-probe coagulation was required to control the blood loss.

The day after the gastroscopy, the patient developed diffuse abdominal pain and cloudy peritoneal effluent. An computed tomography scan of the abdomen showed no visceral perforation or intra-abdominal abscess. Acute bacterial peritonitis was diagnosed based on cloudy peritoneal fluid with a leukocyte count of 11,500 cells/μL consisting of 98% neutrophils. Gram staining of the dialysate was negative. Peritoneal effluent glucose was 214 mg/dL, albumin was 0.4 g/dL, lactic dehydrogenase was 1026 U/L, and amylase was 24 U/L. The patient had a white blood cell count of 16,500/mm³. He was given ceftazidime 1 g intraperitoneally and vancomycin 1 g intravenously. A peritoneal fluid culture was reported positive for *S. viridans* sensitive to β-lactams and macrolides. Vancomycin was discontinued, and cefazidime was given for 14 days, followed by clarithromycin 500 mg daily for 7 days. Peritoneal fluid cultures became negative, and the patient recovered successfully. No relapse or new peritonitis episodes were reported.

Two months later, the patient underwent laparoscopic right nephrectomy, and the biopsy showed a renal-cell carcinoma confined to the kidney. After surgery, the patient developed a retroperitoneal leak requiring transfer to hemodialysis for 3 weeks. He had a new syncope, and a cerebral magnetic resonance imaging scan revealed an arteriovenous malformation complicated by intracerebral hemorrhage. The patient returned to APD uneventfully. Ten months later, he died of pulmonary tuberculosis.

**Discussion**

Upper gastrointestinal endoscopy has been associated with bacteremia and infectious complications such as endocarditis, meningitis, and peritonitis. Variceal sclerosis and esophageal stricture dilatation have the highest risk for bacteremia; lower-risk procedures include biopsy and polypectomy (2). During gastroscopy with or without biopsy, bacteremia ranges in frequency from 0% to 8% (1), usually lasts less than 30 minutes, and is not associated with any infectious complications (7).

In a recent review of iatrogenic peritonitis associated with invasive procedures in continuous ambulatory PD patients, Fried and colleagues reported several peritonitis episodes after colonoscopy (with or without polypectomy), percutaneous endoscopic gastrostomy, barium enema, hysteroscopy, and dental procedures (4). Endoscopic procedures may produce peritonitis through transmural migration of bacteria across the bowel wall or by transient bacteremia that could seed the peritoneum. The composition of PD solutions adversely affects peritoneal host defense and may contribute to the development of peritonitis. The viability of mesothelial cells, peritoneal macrophages, polymorphonuclear leukocytes, and monocytes is depressed. The production of inflammatory cytokines and chemoattractants by those cells is markedly affected. When peritoneal macrophages, polymorphonuclear leukocytes, and monocytes are exposed to dialysis solutions, phagocytosis, respiratory burst, and bacterial killing are diminished (8). Additionally, the concentrations of immunoglobulin G, complement, and fibronectin are reduced when dialysis fluid is present in the peritoneal cavity (9).

Ours is the first reported case of peritonitis after gastroscopy in a patient undergoing PD. An interesting aspect of this case is the association of injection sclerotherapy and heat-probe coagulation used for treating an actively bleeding duodenal ulcer.

Two retrospective series reported peritonitis following percutaneous endoscopic gastrostomy (PEG) in PD patients. Feeding by PEG was associated with a high frequency of fungal peritonitis, dialysate leakage, exit-site infection, transfer to hemodialysis, and peritonitis-related death (10,11). Because of the unacceptably high rate of PD complications with PEG feeding, PD remains a relative contraindication to that procedure (12).

Although *S. viridans* peritonitis has been reported after invasive procedures in continuous ambulatory PD patients, those cases occurred after dental procedures (4,5,13). For PD patients undergoing endoscopic procedures or other invasive interventions, some authors advocate the use of antibiotic prophylaxis, although no supporting data from prospective trials exist (4,14–16).

Current guidelines recommend antibiotic prophylaxis for endoscopic procedures if, as a consequence of immunosuppression or neutropenia, the patient is
at high risk of endocarditis or of symptomatic bacte-

Conclusions

Acknowledgment

References

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