Peritoneal dialysis-related peritonitis with *Klebsiella* pneumoperitoneum mimicking viscus perforation

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Case Report:

A 56-year-old Taiwanese man with end-stage renal disease secondary to diabetes mellitus was on continuous ambulatory peritoneal dialysis (PD) therapy for 3 years. The patient managed PD well (Fig 1A and 1F) by himself, and he experienced no previous episodes of PD-related infection. He was admitted because of abdominal pain, fever up to 38°C, and a cloudy dialysate of 2 days duration. Abdominal examination indicated rebound tenderness and a soft abdomen. The dialysis effluent was turbid; the white count was 37,980/μL with 90% neutrophils. Laboratory data revealed a leukocytosis of 12,110/μL with 85% neutrophils. The standing (Fig 1B) and left lateral decubitus (Fig 1C) chest radiographs showed free air under the right hemidiaphragm. The patient denied having undergone abdominal surgery or intervention except for implantation of a Tenckhoff catheter prior to initiation of PD.

We suspected PD-related peritonitis and began intraperitoneal antibiotic therapy with cefazolin and ceftazidime. However, a small amount of subphrenic free air alerted us to the potential of viscus perforation. Endoscopy indicated the absence of a peptic ulcer. Noncontrast computed tomography (CT) of the abdomen (Fig 1D) indicated free air above the liver (pneumoperitoneum) and no liver abscess. Oral administration of contrast medium followed by CT scanning indicated no evidence of contrast leakage from the gastrointestinal tract. A peritoneal effluent culture yielded *Klebsiella pneumonia*. We changed the treatment to intraperitoneal single-line ceftazidime. The free air completely disappeared on the 7^{th}-day (Fig 1E) imaging and remained absent on the 6^{th}-month (Fig 1F) follow-up imaging, and the dialysate was clear. We considered infection by *K. pneumonia* to be the cause of the PD-associated peritonitis and pneumoperitoneum.

Pneumoperitoneum usually indicates viscus perforation, necessitating emergent laparotomy. However, pneumoperitoneum occurs in one third of PD patients, and its clinical significance is controversial. The causes of pneumoperitoneum include exchange technique error, recent abdominal interventions, visceral perforation, and peritonitis. The first two causes can be easily excluded. However, when pneumoperitoneum occurs with peritonitis in PD patients, viscus perforation can be difficult to distinguish from PD-related peritonitis. This is because viscus perforation and PD-related peritonitis have the same clinical signs, and both may or may not present with subphrenic free air. One paper reported that the incidence of free air was significantly greater in PD patients with peritonitis and viscus perforation than in those with PD-related peritonitis without perforation. However, the amount of free air is not a reliable

diagnostic sign. Therefore, concurrent pneumoperitoneum and peritonitis in PD patients calls for aggressive but prudent evaluation for viscus perforation so as to avoid unnecessary surgery. Contrast radiography can document the location of the perforation, and in our case confirmed the absence of viscus perforation.

One study indicated that there was no significant difference in the prevalence of organisms causing peritonitis in PD patients with and without pneumoperitoneum and that *Staphylococcus aureus* followed by *Staphylococcus epidermidis* dominated over gram-negative organisms. However, while causing peritonitis, *Klebsiella pneumonia* may lead to pneumoperitoneum that can mimic hollow organ perforation, or be trivial, like our case. Nephrologists should be alert to nonsurgical causes of concurrent pneumoperitoneum and peritonitis in PD patients, and should carefully weigh the decision to perform laparotomy, as judged by culture reports and therapeutic response.