Peritoneal Dialysis-Related Peritonitis with *Klebsiella* Pneumoperitoneum Mimicking Viscus Perforation

Editor:

A 56-year-old Taiwanese man with end-stage renal disease secondary to diabetes mellitus had been on continuous ambulatory peritoneal dialysis (PD) therapy for 3 years. The patient managed PD well [Figure 1(A,F)] by himself, and he had to that point experienced no episodes of PD-related infection.

He was admitted because of abdominal pain, fever up to 38°C, and cloudy dialysate of 2 days' duration. Abdominal examination indicated rebound tenderness and a soft abdomen. The dialysis effluent was turbid; the white cell count was 37 980/μL, with 90% neutrophils. Laboratory data revealed leukocytosis of 12 110/μL with 85% neutrophils. Chest radiographs in the standing [Figure 1(B)] and left lateral decubitus [Figure 1(C)] positions showed free air under the right hemidiaphragm. The patient denied having undergone abdominal surgery or any intervention except for implantation of a Tenckhoff catheter before 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. Non-contrast computed tomography imaging of the abdomen [Figure 1(D)] indicated free air above the liver (pneumoperitoneum) and no liver abscess. Oral administration of contrast medium followed by computed tomography imaging showed no evidence of contrast leakage from the gastrointestinal tract. A peritoneal effluent culture yielded *Klebsiella pneumoniae*. We changed the treatment to intraperitoneal single-line ceftazidime. The free air completely disappeared on the day 7 imaging [Figure 1(E)] and remained absent at month 6 follow-up imaging [Figure 1(F)]. Effluent

Figure 1 — Imaging studies: (A) Standing chest radiograph taken 6 months before the reported admission shows an absence of free air. On admission, chest radiographs in the (B) standing and (C) left lateral decubitus views and (D) abdominal computed tomography without contrast show a small rim of free air under the right hemidiaphragm (arrows). After treatment, (E) the free air disappears on day 7 imaging, and (F) remains absent 6 months later.

was clear. We considered infection with *K. pneumoniae* 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 (1), and its clinical significance is controversial (2). The causes of pneumoperitoneum include exchange technique error (1–5), a recent abdominal intervention (1,2), visceral perforation (1–3,6), and peritonitis (1). The first two causes can easily be excluded. However, when pneumoperitoneum occurs with peritonitis in PD patients, viscus perforation can be difficult to distinguish from PD-related peritonitis 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 (7). However, the amount of free air is not a reliable diagnostic sign (1). Concurrent pneumoperitoneum and peritonitis in PD patients therefore calls for aggressive but prudent evaluation for viscus perforation so as to avoid unnecessary surgery. Contrast radiography can document the location of the perforation (7) and, in our case, confirmed the absence of viscus perforation.

One study (1) 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 *S. epidermidis*, dominated over gram-negative organisms. However, while causing peritonitis, *K. pneumoniae* might lead to pneumoperitoneum that can mimic hollow-organ perforation (8) or that can be trivial, as in our case.

Nephrologists should be alert to nonsurgical causes of concurrent pneumoperitoneum and peritonitis in PD patients, and they should carefully weigh the decision to proceed to laparotomy, as judged by culture reports and therapeutic response.

DISCLOSURES

The author has no financial conflict of interest to declare.

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