morganii. We would like to add our recent experience with the same pathogen causing a different clinical course, which we believe might shed new light on the observations by our colleagues.

A 53-year-old white man with end-stage renal disease because of immunoglobulin A nephropathy and on peritoneal dialysis (PD) for 10 months was admitted with abdominal pain and cloudy dialysate. He was afebrile, but his abdomen was tender. Effluent showed an elevated white cell count. Empiric initial treatment consisted of intraperitoneal vancomycin and ceftazidime. Cultures yielded *M. morganii*, sensitive to ceftazidime.

Despite clinical improvement, effluent leukocytes declined only slowly. Ultrasonography was suggestive of left-sided diverticulitis. Computed tomography corroborated that finding, but showed no evidence of abscess formation (Figure 1).

During the subsequent week, C-reactive protein reached a second peak (104 mg/L), and the patient's leukocyte count remained elevated. Despite the patient's stable clinical condition, failure to clear the infection necessitated explantation of the catheter on day 13. Rapid and sustained improvement of inflammatory markers followed. Interim hemodialysis was initiated using a central line. Four weeks later, before catheter re-implantation, repeat computed tomography and colonoscopy showed diverticular disease without evidence of residual inflammation. After 4 months back on PD, this patient continues to do well.

More on Peritonitis by *Morganella morganii*

Editor:

We read with interest the case report by Tsai *et al.* (1) about a woman with peritonitis caused by *Morganella*

Figure 1 — Computed tomography of the abdomen, showing a left-sided colonic diverticulum (arrow), with surrounding bowel-wall thickening and fat stranding, in keeping with diverticulitis.

Current guidelines advocate catheter removal on day 5 of refractory peritonitis (2), but as was stated earlier, our patient's condition was remarkably good, and his effluent cell count was smouldering on a low, albeit abnormal, level. An earlier study showed the safety of conservative management in presumed endogenous peritonitis (3). However, with the benefit of hindsight, we concede that the catheter should have been explanted earlier. All the same, the outcome was ultimately favorable.

In line with the report by Tsai *et al.* (1), peritonitis with *M. morganii* was monobacterial in our patient. That finding notwithstanding, we had substantiated radiologic evidence for peritonitis secondary to diverticulitis. We would like to emphasize that polymicrobial infection is not a prerequisite for secondary peritonitis in the PD population. In a report by Harwell *et al.* (4), 22 of 26 confirmed cases of peritonitis related to visceral injury were monomicrobial. Given the notoriously poor performance of abdominal computed tomography in this population and the disproportionately high mortality in gram-negative peritonitis (5,6), we caution against overreliance on negative imaging results in patients who fail to improve despite appropriate antimicrobial therapy. We acknowledge the more benign course in the case reported by our colleagues, but we wonder whether unrecognized colonic disease was present, accounting for 2 episodes of gram-negative peritonitis within 2 months.

To summarize, our observation adds more details with respect to the emerging role of *M. morganii* as a cause of peritonitis in the PD population, with diverticulitis being the cause in our patient, ultimately resulting in catheter loss.

DISCLOSURES

The authors have no financial conflicts of interest to declare.

M. Windpessl^{1*}

W. Prammer²

R. Asböck³

M. Wallner¹

Division of Nephrology¹

Fourth Department of Medicine

Institute of Microbiology²

Institute of Radiology³

Klinikum Wels-Grieskirchen

Wels, Austria

*email: martin.windpessl@gmx.net

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