

Response to Zhou et al.

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Am J Gastroenterol <https://doi.org/10.1038/s41395-018-0391-2>

To the editor: We are grateful to Drs. Zhang, Wu, Zhou and Xu for their additional analysis of our data with their well-performed cumulative meta-analysis. With the findings that their analysis confirms our findings, particularly with regard to intestinal metaplasia and dysplasia, it helps us to reinforce the need for careful inspection and sampling of Barrett's mucosa before and early after radiofrequency ablation to rule out the presence of prevalent lesions. This cumulative analysis also notes, however, that clear proof that high grade dysplasia (HGD)/cancer detection within the first year of endoscopic therapy is not equal to or greater than that found in subsequent endoscopies is also important. Nevertheless, the practical point is that, even if the rates of developing HGD/cancer in the first year are close to that of subsequent years, it is more than enough reason to apply the principle that early and careful endoscopic follow-up after ablation is performed to find undetected advanced lesions. Whether our data and this additional analysis will lead to guideline changes merits further exploration.

CONFLICT OF INTEREST

Guarantor of the article: David A. Katzka, MD.

Specific author contributions: T.S. and D.A.K. wrote the letter.

Financial support: None.

Potential competing interests: D.A.K.: pharmaceutical trial with Shire. The other author declares that he has no conflict of interest.

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Chronic Diarrhea Related to Colonic Malakoplakia Successfully Treated with Budesonide in a Kidney Transplant Recipient

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Am J Gastroenterol <https://doi.org/10.1038/s41395-018-0382-3>

Dear Editor: A 51-year-old female was hospitalized because of chronic recurrent diarrhea with worsening symptoms. She described watery, bloodless diarrhea of 6–12 times per day for two months. The medical history revealed kidney transplant surgery that was performed eleven years earlier and accompanying hypertension. The medications used by the patient on admission were as follows: mycophenolate mofetil (1000 mg/day), tacrolimus (2 mg/day), prednisolone (5 mg/day), losartan (100 mg/day), carvedilol (12.5 mg/day), and lansoprazole (30 mg/day). The vital signs and systemic examination were normal. The laboratory investigations showed; Hemoglobin: 9 g/dL (normal range [NR]: 11.7–15.5), leukocyte: $4.8 \times 10^9/L$ (NR: 4.1–11.2), neutrophil: $3.2 \times 10^9/L$ (NR: 1.8–6.4), platelet: $203 \times 10^9/L$ (NR: 159–388), alanine-transaminase: 9 U/L (NR: 0–50), aspartate-transaminase: 9 U/L (NR: 0–50), creatinine: 1.9 mg/dL (NR: 0.6–1.1), albumin: 3.54 g/dL (NR: 3.5–5.2), C-reactive protein: 0.22 mg/dl (NR: 0–0.8), and fecal calprotectin: < 30 (NR: 0–30). No red blood

cells or polymorphonuclear leukocytes were seen in stool microscopy and the stool culture was negative as well as tests for clostridium difficile and celiac disease. The esophagogastroduodenoscopy was unremarkable except for mild gastritis. Colonoscopy revealed yellowish-white, flat elevated lesions with patchy involvement of the ascending colon (Fig. 1). The histopathological examination of the biopsy of the lesions was consistent with colonic malakoplakia (Fig. 2). In the random biopsies taken of normal-appearing mucosa of the ascending colon, descending colon and rectum, the absence of microscopic colitis or amyloidosis was documented in the histopathological examination. The immunosuppressive medications were modified (Mycophenolate mofetil was stopped, tacrolimus dose was decreased to 1 mg/day, and azathioprine 75 mg/day was initiated), but the diarrhea continued during the subsequent three weeks. Budesonide 9 mg/day was started for diarrhea and the tacrolimus dose was increased to 2 mg/day as ordered by the consultant nephrologist. At the end of the third week of budesonide treatment, the patient had improved dramatically and was symptom-free. Budesonide treatment was continued for three more months and then stopped gradually. The diarrhea symptoms did not recur during the nine-month follow-up after treatment cessation.

Malakoplakia is a chronic inflammatory disorder that can affect various parts of the body, including the urinary tract, gastrointestinal tract, lymph nodes, lung, brain, pancreas, bone, and skin. The gastrointestinal system is the second most common site of involvement of the disease after the urinary tract, and the colon and rectum are predominantly affected. Although the pathogenesis is not completely known, gram-negative bacterial infections, especially *Escherichia coli*, and altered inflammatory activation have been considered responsible in the disease etiology. Malakoplakia is usually encountered with comorbid conditions such as malignancies, organ transplantation, sarcoidosis, tuberculosis, liver diseases, ulcerative colitis, malnutrition, mycotic infections, and immunodeficiency syndromes [1, 2].