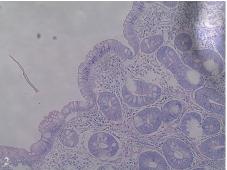
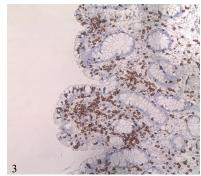
Ileal Villous Atrophy in a Hypertensive Patient: Guess What?

Vincent Zimmer^{1,2}, Christoph Heinrich³

1) Department of Medicine, Marienhausklinik St. Josef Kohlhof, Neunkirchen; 2) Department of Medicine II, Saarland University Medical Center, Saarland University, Homburg; 3) Institute of Pathology Saarbrücken-Rastpfuhl, Saarbrücken, Germany







An 81-year-old female was referred for ileocolonoscopy after an abdominal computed tomography scan suggested minor questionable wall thickening in the right hemicolon. More important, the patient reported chronic diarrhoea passing up to 8 watery stools per day for about six months in conjunction with fluctuating right lower quadrant pain. Medical history was significant for arterial hypertension under 8 mg candesartan and hydrochlorothiazide 12.5 mg. Lower gastrointestinal endoscopy indicated advanced villous atrophy, scalloping of folds, nodularity and mosaic pattern in the terminal ileum as highlighted by high-quality underwater endoscopy (Fig. 1). This was confirmed by histopathology demonstrating incomplete villous atrophy and intraepithelial lymphocytosis up to 25/100 epithelial cells on light microscopy (Fig. 2, H&E staining, 20x) and dedicated immunohistochemistry (CD3) (Fig. 3, 20x). Intriguingly, the patient reported rapid and complete normalization of stool frequency and consistency within 5 days after discontinuation of candesartan medication. An ancillary celiac disease serology [transglutaminase immunoglobulin A (IgA) antibodies and serum IgA] proved unremarkable. The patient was initially advised to undergo esophago-gastroduodenoscopy to evaluate for architectural changes in the duodenum, which, however, was refused, as was repeat ileocolonoscopy to formally assess for pathological remission.

Olmesartan-associated enteropathy has first been described by the group of Rubio-Tapia et al. [1] from the Mayo Clinic in Rochester in a population of individuals referred for work-up of presumed sero-negative celiac disease, thus introducing sartan enteropathy as a clinically relevant differential diagnosis of villous atrophy. Later case series and individual reports have extended the spectrum of gastrointestinal manifestations to villous atrophy of the more distal parts of the small bowel and microscopic colitis [2]. While olmesartan is typically associated with sartan-induced enteropathy other angiotensin II receptor blocker (ARBs), including candesartan, have been reported in the literature. The specific mechanism behind sartan-associated enteropathy remains to be better delineated. However, cell-mediated immune damage and inhibitory effects of gastrointestinal-expressed angiotensin II receptors on transforming growth factor- β (TGF- β), dysbalancing cellular gut homeostasis, are being discussed [3].

Taken together, to avoid undue clinical decisions, clinicians and/or endoscopists should remember ARB-induced enteropathy and its wide spectrum as an important differential of diarrhoea with rapid and durable remission after stopping the drug as was the case in the presented patient.

Corresponding author: Vincent Zimmer, vincent.zimmer@gmx.de

Conflicts of interest: None to declare.

REFERENCES

- 1. Rubio-Tapia A, Herman ML, Ludvigsson JF, et al. Severe spruelike enteropathy associated with olmesartan. Mayo Clin Proc 2012;87:732-738. doi:10.1016/j.mayocp.2012.06.003
- Zimmer V, Heinrich C. Olmesartan-induced lymphocytic colitis: a specific gap in the guideline? Clin Res Hepatol Gastroenterol 2021;45:101644. doi:10.1016/j.clinre.2021.101644
- Choi EY, McKenna BJ. Olmesartan-Associated Enteropathy: A Review of Clinical and Histologic Findings. Arch Pathol Lab Med 2015;139:1242-1247. doi:10.5858/arpa.2015-0204-RA