Editorial: proton pump inhibitors (PPIs) and microscopic colitis

Microscopic colitis is a common cause of chronic, nonbloody diarrhoea that has a large influence on health-related quality of life and cost of care. Depending on patients’ age, microscopic colitis can be diagnosed in 10%-30% of the patients presenting with chronic, nonbloody diarrhoea.1 Two main entities are identified: collagenous; and lymphocytic colitis, comprising 36% and 64% of cases, respectively.2

The awareness for microscopic colitis has risen over the last decade following an increase in incidence rates from 4.6 to 24.7 per 100,000 person-years.3 Unfortunately, therapeutic options are scarce; the only evidence-based treatment option is budesonide microspheres.4

The development of microscopic colitis have been reported including smoking, NSAIDs and proton pump inhibitors (PPIs). A large nationwide registry study from Denmark investigated the relation between PPI use and microscopic colitis more closely.6

Bonderup et al identified 10,652 patients with first-diagnosed microscopic colitis between 2004 and 2013. They confirmed earlier reported strong associations7 between current PPI use and microscopic colitis incidence with an adjusted odds ratio (OR) for collagenous colitis of 6.98 (95% CI: 6.45-7.55) and for lymphocytic colitis of 3.95 (95% CI: 3.60-4.33). Some interesting issues still remain for future investigation. First, cases were included up to 2013. Expanding this cohort with more recent inclusions, and performing subgroup analysis comparing the earlier years with more recent years to investigate presence of a cohort effect, would have been interesting. Specifically, the indication for PPIs changed over time from treatment of gastric-acid related symptoms and functional dyspepsia to peptic ulcer prophylaxis with PPIs. The influence of NSAIDs combined with PPIs still needs to be studied with either medication separately. By studying the effect of H2RA agents, a possible class effect of PPIs can be ruled out and a first step into establishing a causal relationship between acid inhibition and microscopic colitis can be assessed.

The pathophysiological explanation for PPI-induced microscopic colitis remains unclear. Therefore, the lack of a dose-response relation is of the utmost interest. Until this paper was published, this relation was not investigated.7 A comparison of 3,737 cases of microscopic colitis with and without current PPI use showed no increased risk of microscopic colitis with increasing PPI dose.6 The lack of a dose-response relation suggests an idiosyncratic adverse

References


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reaction. Accordingly, stopping the PPI will possibly decrease the symptoms within days.

Although the relation between PPIs and microscopic colitis has been known for many years, the effect of stopping the PPI on these symptoms has not been well investigated. A well documented cohort of patients, where PPIs will be discontinued after the diagnosis of microscopic colitis could be the missing link between the epidemiologic association between PPIs and microscopic colitis and clinical practice.

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ORCID

Robin Krol http://orcid.org/0000-0001-5655-9725
Martijn G. H. van Oijen http://orcid.org/0000-0003-4581-1440

LINKED CONTENT

This article is linked to Bonderup et al paper. To view this article visit https://doi.org/10.1111/apt.14916.

REFERENCES