Letter to Editor

Lymphocytic gastritis and celiac disease

Angelo Viscido, Giovanni Latella

Gastroenterology Unit. Department of Life, Health and Environmental Sciences. University of L'Aquila, L'Aquila, Italy

To the Editor,

We read with great interest the review article "Lymphocytic gastritis" by Puderecki et al., which was recently published in your journal [1]. The article describes the features of lymphocytic gastritis (LG), a rare form of gastritis with unclear pathogenesis.

The diagnosis of LG is based on histology which reveals intraepithelial lymphocytosis (> 25 intraepithelial lymphocytes per 100 gastric surface and foveolar epithelial cells). The endoscopic appearance of LG can vary from normal mucosa to aphthous erosions, nodularity, local spots, polyps, and ulcers. The most common locations of the lesions are the body and the antrum. With regard to etiology, Celiac disease (CD) is the main reported cause of LG, followed by *Helicobacter pylori* infection. After a careful review of the argument, Puderecki et al. conclude that there is no one exact cause of LG, and rather than being a separate disease, LG is more likely a sign of the disease with which it is associated [1].

We wrote to remark on the strong connection between LG and CD. Such a connection may allow some etiopathogenetic and clinical speculations.

CD is an autoimmune disease involving the small bowel mucosa triggered by the ingestion of gluten in genetically predisposed subjects. The exclusion of gluten from the diet results in the healing of the mucosa and resolution of intestinal malabsorption [2]. It is noteworthy that in patients with concomitant CD and LG, the exclusion of dietary gluten results in reversal of LG too [3].

LG is frequently present in patients with CD at the time of the diagnosis, being its incidence strictly related to the severity of duodenal lesions, in particular villous atrophy [4]. A prospective study from our Institution showed that up to 16% of patients with CD have LG at the time of diagnosis. After stratification based on the severity of the CD lesions, LG was present in almost 90% of the patients presenting with severe duodenal injury (Marsh 3 grade). Moreover, patients with LG and villous atrophy tend to have a higher antibody count and lower serum albumin levels. Notably, almost all cases of LG improved after a gluten-free diet [3].

These data, according to those presented by Puderecki *et al*., lead to suggest that LG may be one form of presentation of CD. The greater the inflammatory activity in the duodenum, the higher the number of lymphocytes in the stomach. Both conditions improve after a gluten-free diet. From a pathogenetic point of view, it could be argued that LG associated with CD is a gastric immunological response to gluten, identical to that occurring in the duodenum lymphocytes. Furthermore, the observation that CD and LG often share certain HLA alleles strongly supports this hypothesis [1].

From a clinical point of view, we agree with the recommendation of Puderecki et al. that the finding of LG should suggest looking for the presence of CD. Furthermore, the finding of LG in patients with known CD should suggest evaluating adherence to gluten-free diet, as probably the antigen – i.e. gluten – is still acting on lymphocytes.

The authors declare no conflict of interest.

References

1. Puderecki M, Wronecki L, Cięszczyk K, et al. Lymphocytic gastritis. Pol J Pathol 2019; 70: 155-161.
2. Ciccone A, Gabrieli D, Cardinale R, et al. Metabolic alterations in celiac disease occurring after following a gluten-free diet. Digestion 2019; 100: 262-268.
3. Gabrieli D, Ciccone F, Capannolo A, et al. Subtypes of chronic gastritis in patients with celiac disease before and after gluten-free diet. United European Gastroenterol J 2017; 5: 805-810.

4. Feeley KM, Heneghan MA, Stevens FM, et al. Lymphocytic gastritis and coeliac disease: evidence of a positive association. J Clin Pathol 1998; 51: 207-210.

Address for correspondence

Angelo Viscido
Department of Life, Health and Environmental Sciences
University of L'Aquila
L'Aquila, Piazzale Salvatore Tommasi 1
L'Aquila 67100, Italy
tel. +39 086 243 4746
e-mail: angelo.viscido@univaq.it