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Mixed-etiology atrophic gastritis: from overlap label to practical risk assessment

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I read with great interest the review by Rokkas and Rugge on the malignant potential of atrophic gastritis arising from different etiologies [1]. The authors distinguish *Helicobacter pylori* (*H. pylori*)-associated atrophic gastritis (AG) from autoimmune AG and also discuss patients with features of both [1]. This mixed-etiology group needs a practical definition, because the term is currently applied to clinically different situations.

Patients may have evidence of current or previous *H. pylori* injury together with corpus-predominant atrophy, anti-parietal cell or anti-intrinsic factor antibodies, hypergastrinemia, pernicious anemia, or enterochromaffin-like cell hyperplasia. These findings may reflect concurrent infectious and immune-mediated injury rather than a simple overlap of labels. However, available prospective data do not support a routine 1-2-year surveillance interval for all such patients [1].

At minimum, evaluation should document current and previous *H. pylori* status, the distribution of atrophy, relevant antibodies, fasting gastrin, pepsinogen I and the pepsinogen I/II ratio, pernicious anemia, and enterochromaffin-like cell hyperplasia. OLGA/OLGIM staging should be based on high-quality endoscopy and Sydney-protocol biopsies [1,2]. These data can distinguish true coexistence from isolated autoimmune gastritis, remote *H. pylori* injury, or nonspecific atrophy.

Surveillance should follow the most important established risk feature: extensive or incomplete intestinal metaplasia, OLGA/OLGIM stage III-IV, dysplasia, family history of gastric cancer, severe corpus atrophy, marked hypergastrinemia, enterochromaffin-like cell hyperplasia, or previous type 1 gastric neuroendocrine neoplasia [2,3].

Microbiome profiling, epigenetic biomarkers, liquid biopsy, and artificial intelligence-assisted endoscopy remain investigational. Current management still depends on endoscopic inspection, systematic biopsies, histologic staging, and careful etiologic assessment. Prospective studies should determine whether concurrent *H. pylori*-related and autoimmune injury increases the risk of adenocarcinoma or gastric neuroendocrine neoplasia beyond either condition alone.

Disclosure

In the course of preparing this work, the author utilized a generative AI tool to enhance and refine the basic text. Following this, the author conducted a thorough review and made necessary edits, thereby assuming full responsibility for the originality, scientific integrity, and final wording of the publication.

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