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Enterochromaffin-like Cell Hyperplasia as Identification Marker of Autoimmune Gastritis in Patients With *Helicobacter pylori* Infection in the Context of Gastric Premalignant Lesions

To the Editor:—With interest, we read the article "Features That Aid Identification of Autoimmune Gastritis in a Background of Active *Helicobacter pylori* Infection" by Choudhuri et al.[1] The authors found that full-thickness oxyntic mucosa inflammation combined with oxyntic gland loss and enterochromaffin-like (ECL) cell hyperplasia may help identify patients with autoimmune gastritis (AIG) with concurrent *Heliobacter pylori* infection from *H pylori*–associated gastritis. In a retrospective analysis, 6 of 7 *H pylori*–positive AIG cases showed loss of oxyntic glands and ECL hyperplasia. In a prospective cohort, oxyntic gland loss was seen in 10 of 11 *H pylori*–positive AIG cases, with ECL hyperplasia present in 8 cases. No oxyntic gland loss or ECL hyperplasia was seen in 8 controls, either before or 10 years after *H pylori* eradication, and thus a more careful pathologic examination of biopsies taken from *H pylori*–positive individuals may allow a better diagnosis for and earlier treatment of AIG patients.

These findings are of importance because recently the management of epithelial precancerous conditions and lesions of the stomach (MAPS 2019) guidelines have indicated that in addition to intestinal metaplasia (IM) and gastric atrophy (GA), AIG diagnosis warrants surveillance of patients for early detection of gastric cancer.[2] Several studies have suggested that AIG development is associated with *H pylori* infection,[3] which is also the main risk factor for IM and gastric cancer development. Interestingly, in the cohort of Choudhuri et al,[1] 51% of AIG patients presented with concomitant IM. However, none of the controls were IM positive. With a clear link between *H pylori*, GA, IM, and AIG, it is of importance to know whether the defined pathologic findings also distinguish AIG in *H pylori*–positive individuals in a background of premalignant lesions. To this end, we examined a cohort of patients who received a diagnosis of gastric precancerous lesion (GPL), including GA and/or IM. Based on pathology availability, 7 men and 15 women from the PROREGAL study, with a mean age of 56 ± 12 years, were included for retrospective analysis.[4] All patients received a diagnosis of GPL by gastroendoscopy, and serum was collected at baseline endoscopy. The presence of anti–parietal cell antibodies was determined by indirect immunofluorescence antibody test and H^+K^+ATPase-specific EliA automated enzyme fluoroimmunoassay. *H pylori* infection was identified based on pathology findings or the presence of anti–*H pylori* antibodies. A total of 9 of 22 cases were identified as having AIG in the context of *H pylori* infection based on both pathology and serum tests (Table). ECL cell hyperplasia was present in 6 of these, and all showed mucosal atrophy in the corpus. A total of 13 cases showed *H pylori*–associated gastritis without AIG, with ECL cell hyperplasia observed in 1 case. How-

Pathologic Features of Study Cases

Feature	Autoimmune Gastritis		*H pylori*–Associated Gastritis	
	Active *H pylori* Infection (n = 2), No. (%)	Previous *H pylori* Infection (n = 7), No. (%)	Active *H pylori* Infection (n = 5), No. (%)	Previous *H pylori* Infection (n = 8), No. (%)
Corpus				
Gastritis	2 (100)	3 (43)	5 (100)	3 (38)
Oxyntic gland loss				
Absent	1 (50)	5 (71)	4 (80)	8 (100)
≤50%	1 (50)	2 (29)	1 (20)	0
≥50%	0	0	0	0
Atrophy				
Mild	0	1 (14)	0	0
Moderate	0	1 (14)	1 (20)	0
Severe	0	5 (71)	0	1 (13)
ECL cell hyperplasia				
Linear	0	5 (71)	1 (20)	0
Linear and nodular	0	1 (14)	0	0
Intestinal metaplasia				
Mild	0	0	0	0
Moderate	1 (50)	2 (29)	0	0
Severe	0	4 (57)	0	1 (13)
Antrum sample				
Gastritis	2 (100)	2 (29)	4 (80)	4 (50)
Atrophy				
Mild	0	0	0	1 (13)
Moderate	0	0	2 (40)	3 (38)
Severe	2 (100)	0	1 (20)	2 (25)
Intestinal metaplasia				
Mild	0	0	1 (20)	2 (25)
Moderate	1 (50)	0	1 (20)	3 (38)
Severe	1 (50)	0	1 (20)	3 (38)

Abbreviation: ECL, enterochromaffin-like.

ever, the serologic results of this patient indicated near-positive anti–parietal cell antibody levels, suggesting that patient was in end-stage AIG.[5] These data confirm that ECL cell hyperplasia is a prevalent manifestation in AIG, even in patients with previous *H pylori* infection and already established GPL. Awareness should be raised among clinicians and pathologists to look for AIG in the context of GPL, which may be facilitated by the assessment of ECL hyperplasia in these patients.

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In Reply.—We thank Drs Guo, Spaander, and Fuhler for their interest in our article; we were pleased to learn of their data related to autoimmune gastritis (AIG) in the setting of ongoing or remote *Helicobacter pylori* infection.[1] Similar to our findings, these authors found that enterochromaffin-like (ECL) cell hyperplasia is a relatively specific and sensitive marker of AIG.

We wish to highlight 2 aspects of the work by Guo et al that are complementary to our findings. First, their AIG–*H pylori* cohort comprises anti–parietal cell antibody (APCA)–positive cases. These antibodies are present in the 80% to 90% of patients with AIG; thus, their presence supports the histologic diagnosis, as the pathologic features do overlap somewhat with those of longstanding *H pylori* infection.[2,3] Serologic studies for AIG-associated antibodies were performed (and were positive) in only 3 of our cases. Using an additional line of evidence to diagnose AIG strengthens the conclusion that ECL cell hyperplasia is a reliable marker for this disorder. Second, inclusion criteria for our *H pylori* control group specified that cases would have at least 10 years of follow-up and would be *H pylori*–negative at follow-up. As Drs Guo, Spaander, and Fuhler point out, no intestinal metaplasia was detected in this group. This may have been due to successful eradication therapy, but also likely reflects sampling error in a small cohort (n = 10). Thus, we were not able to determine whether ECL cell hyperplasia was a distinguishing feature between AIG and cases of *H pylori* infection with intestinal metaplasia or extensive atrophy. The authors specifically chose cases that showed atrophy or intestinal metaplasia and arrived at the conclusion that ECL cell hyperplasia is equally reliable in this setting.

In summary, the findings of Guo et al are in agreement with our recent work and further clarify the diagnostic features of AIG in various settings. These findings are clinically important, particularly in light of recent clinical surveillance guidelines.[4]

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Getting Pathologists Out From Behind the Paraffin Curtain: How the College of American Pathologists Began Its Community Outreach and Membership Media Training

To the Editor.—During a spring 1981 College of American Pathologists (CAP) Governor's Meeting, Pierre Keitges, MD, and Dan Seckinger, MD, asked me to discuss the feasibility of starting a CAP Public Outreach program. In 1980 I had started the Pima County Medical Society Media Committee and a weekly Tucson half-hour radio AM talk show that discussed commonplace medical challenges—everything from prematurity to hemorrhoids. Public call-in questions added listener engagement.

Keitges and Seckinger pitched the concept to the rather skeptical Board of Governors, convincingly arguing, "The public doesn't know about pathology and what we add to care" and "Most doctors don't see pathologists are real physicians," and they won over the board, which awarded a small trial allotment. So, the CAP Regional Communication Advocacy Committee was born in 1981, and, later, the CAP Regional Public Service Committee was born in 1984. William Kuehn, PhD, an ex-Jesuit priest and a public relations professional, was hired to