Incomplete Intestinal Metaplasia is Rare in Autoimmune Gastritis

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Background and Hypothesis

There are two histologic subtypes of intestinal metaplasia (IM). Complete type, which is intestinal metaplasia that resembles small intestinal mucosa with Paneth cells and a microvillous brush border, and incomplete type, that resembles colonic mucosa and lacks Paneth cells and a microvillous brush border. Incomplete intestinal metaplasia is associated with greater gastric cancer risk versus complete intestinal metaplasia and the AGA guidelines (1), along with several other international guidelines, recommend including IM type in pathology reports. In a long-term follow-up of a large cohort of patients with *Helicobacter*-naïve autoimmune atrophic gastritis (AIG), we did not detect any increased risk for gastric cancer (2). Therefore, we hypothesized that since incomplete intestinal metaplasia has been shown to be a reliable harbinger of increased gastric cancer risk, it should be uncommon in patients with AIG.

Methods

We assessed the IM type in biopsies from 377 subjects with IM and one of the following gastric phenotypes: 58 subjects with minimal changes gastritis; 135 with reactive gastropathy; 136 with *H. pylori* chronic active gastritis; and 48 with AIG. In an attempt to avoid including patients with previous *H. pylori* infection, we used strict criteria for the diagnosis of AIG, requiring an uninflamed antrum without IM, corpus-restricted atrophy, and ECL-cell hyperplasia. Biopsies were reviewed to confirm diagnosis, location and categorize the type of IM (complete or incomplete). The prevalence of incomplete IM was compared amongst gastritis groups. Differences was evaluated using odds ratios (OR) and their 95% confidence intervals (95% CI).

Results

Incomplete IM was present in: none of 58 patients with normal gastric mucosa; 7/135 patients (5.2%) with reactive gastropathy; 52/136 with *H. pylori* gastritis (38.2%); and in 4/48 with autoimmune gastritis (8.3%).

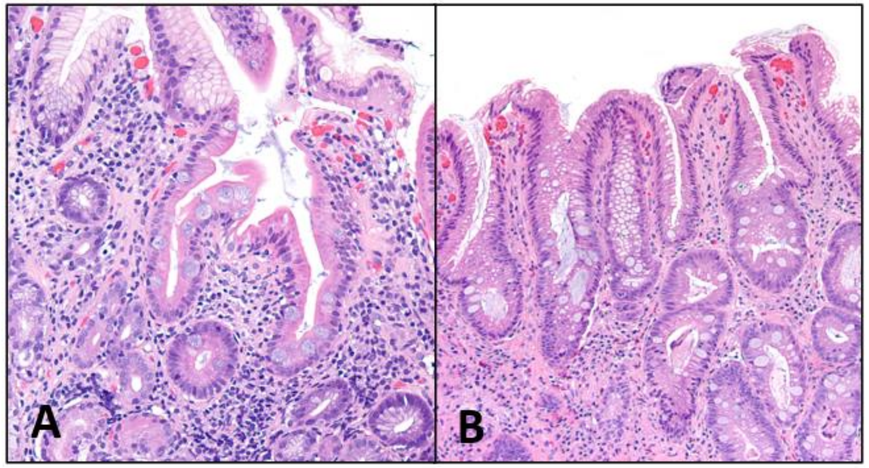

Figure 1. Photomicrographs of complete intestinal metaplasia with more consistently sized and shaped goblet cells and a sharp microvillous brush border (A), incomplete intestinal metaplasia with variable sized and shaped goblet cells and no microvillous brush border (B)

Patients with IM	Total	Incomplete IM
MINIMAL CHANGES	58	0
REACTIVE GASTROPATHY	135	7 (5.2%)
HELICOBACTER GASTRITIS	136	52 (38.2%)
AUTOIMMUNE GASTRITIS	48	4 (8.3%)

Table 1. The prevalence of incomplete intestinal metaplasia in patients with different histologic backgrounds

Conclusion

Compared to subjects with *H. pylori* gastritis – a condition known to carry a high risk of GC – patients with AIG had a low prevalence of incomplete IM, providing further support to the concept that AIG, in the absence of previous or concurrent *Helicobacter* infection, is not associated with a significant GC risk.

References

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