Gastric Intestinal Metaplasia: A Retrospective Analysis in a District General Hospital in the United Kingdom

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Background

Gastric intestinal metaplasia (IM) has been identified as part of a carcinogenesis sequence leading to the development of gastric cancer (figure 1). The largest Western European cohort study found a 10-year incidence of gastric cancer in patients with IM to be 1.8%. Recent evidence-based European Society of Gastrointestinal Endoscopy (ESGE) guidelines have highlighted additional risk factors, such as the intragastric distribution of IM (extensive represents a higher risk than focal IM) and the presence of Helicobacter pylori. Their suggestions include (1) a minimum of 4 biopsies (figure 2) of the proximal and distal stomach, including the greater and lesser curvature, (2) a surveillance strategy in those at particular risk, in order to facilitate earlier cancer diagnosis, and (3) eradication of H. pylori to slow the progression of carcinogenesis.

Methods

Using the keywords “intestinal metaplasia”, the histology database for the Queen Elizabeth Hospital, Woolwich in South East London was reviewed over a 12-year period (2000-2011) to identify patients with IM on gastric biopsy. Patients with IM at the gastrointestinal junction were excluded in view of potential that these could be Barrett’s cases. Their histology and endoscopy reports were scrutinized to elucidate the number and site of biopsies taken and if H. pylori was associated. The terminology used, specifically with regards to “extensive” and “focal” IM, was compared with the suggestions from the ESGE guidelines. We identify patients who might be candidates for surveillance endoscopy. The database of IM patients was cross-referenced with a database of upper gastrointestinal cancer patients over the same period to investigate the development of cancer in patients with IM.

Results

A total of 175 patients with gastric intestinal metaplasia were identified from the pathology database. Of these, seven patients had a confirmed cancer diagnosis subsequent to the index gastric biopsies over the period 2000-2011.

Three gastric adenocarcinoma cases were identified: one in association with IM on index biopsy; another was diagnosed with gastric cancer within two months of the initial IM biopsy, suggestive of a metachronous cancer; only in one patient was gastric intestinal metaplasia demonstrated on biopsy in February 2000 (4 antral-only biopsies taken, “extensive” on report, no H. pylori), who subsequently developed gastric cancer April 2008. Helicobacter pylori were associated with 20/175 (11.4%) of gastric IM biopsies, but was not detected on the biopsies associated with the three cancer cases.

In the four patients where gastric carcinoma was not detected, diagnoses were small cell neuroendocrine carcinoma, poorly-differentiated adenocarcinoma of unknown origin on omental biopsy, duodenal adenocarcinoma (one year after index gastric biopsies) and pancreatic adenocarcinoma.

After review of pathology reports, 37/175 (21.1%) of cases with gastric IM, the pathologist did not receive sufficient clinical information specifying the site of the biopsies. Of those where the biopsy site was specified, only 10/138 (7.2%) had sufficient biopsies (4 biopsies of the proximal and distal stomach) at the index endoscopy. In terms of pathology reporting nomenclature, the use of the phrase “extensive” was used in 27/175 (15.4%) cases, despite either insufficient biopsies being taken to justify use of the term, or the site of the biopsies not being specified. Only 1/175 patient had the recommended number of biopsies and extensive IM.

Conclusion

Gastric intestinal metaplasia is an established precursor to adenocarcinoma. This study identified 175 patients with gastric intestinal metaplasia on endoscopic biopsy over the period 2000-2011, of which one patient subsequently developed gastric adenocarcinoma 8 years later. Since surveillance endoscopy is not routine practice in the Trust, all biopsies were incidental findings.

An extensive distribution of gastric intestinal metaplasia, occurring both in the antrum and corpus, poses a higher risk of progression to adenocarcinoma compared with focal distribution, and warrants 3 yearly surveillance endoscopies according the ESGE guidelines. This study suggests that, whilst in 21.1% of cases enough information specifying the biopsy site was not received by the pathologist, of the remainder only 7.2% had sufficient biopsies (≥ 2 of antrum, ≥ 2 corpus including greater and lesser curvature). Thus, the other cases should have repeat endoscopies with sufficient biopsies to ascertain the intragastric distribution of intestinal metaplasia, to risk stratify and decide on surveillance.

We identified discrepant use of nomenclature in pathology reporting. In 15.4% of cases, the phrase “extensive metaplasia” was used despite either an inadequate number or site of biopsies, or no specification of the site disclosed on the clinical details form submitted to the pathologist, highlighting its inaccurate use. Adoption of the Operative-Link for Gastric Intestinal Metaplasia (OLGIM) tool would rectify this.

Helicobacter pylori is associated with progression of carcinogenesis and the ESGE advocates its eradication. This applied to 11.4% IM biopsies. The three gastric carcinoma cases were not associated with H. pylori. Use of proton pump inhibitors and results of rapid urease testing were not examined.

This study has limitations including a small sample size and insufficient time span of analysis. Nevertheless, it reveals further work is needed to risk stratify and survey this important pre-cancerous condition.

Aims

To review local histological data to identify the prevalence of gastric intestinal metaplasia, the adequacy of numbers of biopsies and terminology used in reporting, association with H. pylori, and the development of gastric cancer in those known to have intestinal metaplasia.

References


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Figure 1. Gastric carcinogenesis cascade

Figure 2. European guidelines recommended biopsy sites include gastric antrum and corpus, lesser and greater curvature, with a minimum of 4 biopsies in total for optimal staging/surveillance.1