

Hyperplastic Polyps of the Stomach

Associations With Histologic Patterns of Gastritis and Gastric Atrophy

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Hyperplastic polyps are common gastric lesions characterized by hyperplastic foveolae with variable amounts of inflamed stroma. Their pathogenesis is unknown, but they have been reported to occur in association with various forms of chronic gastritis, particularly autoimmune gastritis and *Helicobacter pylori* gastritis. Comprehensive histologic evaluation of the background mucosal pathology in patients with hyperplastic polyps has not been previously performed. We studied 160 patients with gastric hyperplastic polyps and characterized endoscopic and histologic features of the polyps (i.e., location, multiplicity, and presence of dysplasia and adenocarcinoma) and the background gastric mucosa (i.e., intestinal metaplasia, dysplasia, carcinoma, and presence and classification of gastritis). Hyperplastic polyps were most common in the antrum (60%) and were multiple in 20% of patients. Focal intestinal metaplasia of the polyp was present in 16% and dysplasia in 4% of patients. Only one patient (0.6%) had adenocarcinoma within the polyp. Evaluation of the surrounding gastric mucosa showed at least focal intestinal metaplasia in 37% of patients, adenoma or low-grade flat epithelial dysplasia in 2%, and synchronous or metachronous adenocarcinoma in 6%. Eighty-five percent of patients had inflammatory mucosal pathology, most commonly active chronic *H. pylori* gastritis (25%), reactive or chemical gastropathy (21%), and metaplastic atrophic gastritis of the autoimmune (12%) or environmental (8%) type. These results indicate a strong association between various forms of gastritis and the development of hyperplastic polyps and further emphasize the importance of biopsy of the nonpolypoid gastric mucosa during endoscopic examination.
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Hyperplastic polyps are among the most common epithelial polyps of the stomach.[1,6,26,37] Found throughout the stomach, they can range from small, innocuous-appearing nodules of a few millimeters to large mass lesions of many centimeters that may be endoscopically mistaken for carcinomas. Histopathologically, hyperplastic polyps are characterized by hyperplastic, elongated, or dilated foveolae that are admixed with variable amounts of inflamed stroma. The lining of the foveolae is composed of mature gastric mucous cells with abundant cytoplasm, except in areas of surface erosions, where nuclear enlargement and depletion of cytoplasmic mucin may lend a markedly regenerative appearance.[26] The prevalence of true dysplasia arising in hyperplastic polyps is debated, but reported rates have varied from 1.9% to 19%.[4,5,10,18,19] Similarly, although cases of adenocarcinoma developing in association with hyperplastic polyps have occasionally been reported,[2,7,11,13,29,30,32,43] larger series of patients with hyperplastic polyps have reported adenocarcinoma rates ranging from 0% to 13.5%,[3,4,6,10,15,18,21,28,31,37,44] with the higher rates generally reflective of carcinomas developing in gastric mucosa outside the polyp.

The pathogenesis of hyperplastic polyps is unclear. Unlike gastric fundic gland polyps, which tend to arise in otherwise normal gastric mucosa,[16,24,34] hyperplastic polyps have been reported in association with various types of chronic gastritis, particularly autoimmune gastritis,[17,22,23,27,39] *Helicobacter pylori* gastritis,[25,28,40] and the postantrectomy stomach.[5,11,20,36] They have also been rarely reported to arise after acute gastric ulceration and after laser therapy for gastric antral vascular ectasia or watermelon stomach.[9,38]

Despite these reported associations with varying forms of chronic and active gastritis, a comprehensive histologic assessment of the background gastric mucosa in patients with hyperplastic polyps has not been previously reported. In particular, the association between gastric

hyperplastic polyps and chemical or reactive gastropathy has not been studied in detail. In this study, we characterized the background gastric mucosal features in a series of 160 patients with hyperplastic polyps with regard to the presence and type of gastritis and the frequency of intestinal metaplasia, dysplasia, and gastric carcinoma in the polyps and in the surrounding gastric mucosa.

MATERIALS AND METHODS

Patient Population and Specimens

Cases were accrued by searching the surgical pathology files at The Johns Hopkins Hospital for gastric specimens in which hyperplastic polyp was reported in the diagnosis between January 1996 and April 2000. Only polyps showing well-developed foveolar hyperplasia with or without increased inflammatory cells in the lamina propria or surface erosions were included in the study (Fig. 1). Polyps showing predominantly inflammatory infiltrate or granulation tissue without elongation, dilatation, or tortuosity of the foveolae (pure inflammatory polyps) were excluded. The study population consisted of 160 patients with at least one gastric hyperplastic polyp. None of the 160 patients was known to have a polyposis syndrome, including juvenile polyposis, Peutz-Jeghers syndrome, or Cronkhite-Canada syndrome, in which the gastric polyps may resemble sporadic hyperplastic polyps. Specimens were obtained from surgical resections in seven patients and during endoscopic examination in 153 patients. Nine cases were seen in consultation, and the rest were from The Johns Hopkins Hospital. Clinical and endoscopic information was obtained from the patients' records or computerized patient files.

Histologic Evaluation

In addition to hematoxylin and eosin stains, all in-house specimens, from 151 patients, were stained with Diff-Quik (Dade Behring, Düdingen, Switzerland) for identification of *H. pylori* and with periodic acid–Schiff and alcian blue at pH 2.5 for evaluation of intestinal metaplasia. Immunohistochemical stains were performed for *H. pylori* (1:200 dilution, Dako, Carpinteria, CA, USA) in nine cases, for cytomegalovirus (1:30 dilution, Dako) in six cases, for chromogranin (1:4,000 dilution, Boehringer-Mannheim, Indianapolis, IN, USA) or gastrin (1:4,000 dilution, Zymed, San Francisco, CA, USA) in the histologic evaluation of autoimmune gastritis in 24 cases, and for CD3 (1:300 dilution, Dako), CD20 (1:200 dilution, Dako), or CD43 (1:400 dilution, Becton Dickinson, San Jose, CA, USA) in the evaluation for suspected mucosa-associated lymphoid tissue lymphoma in five cases.

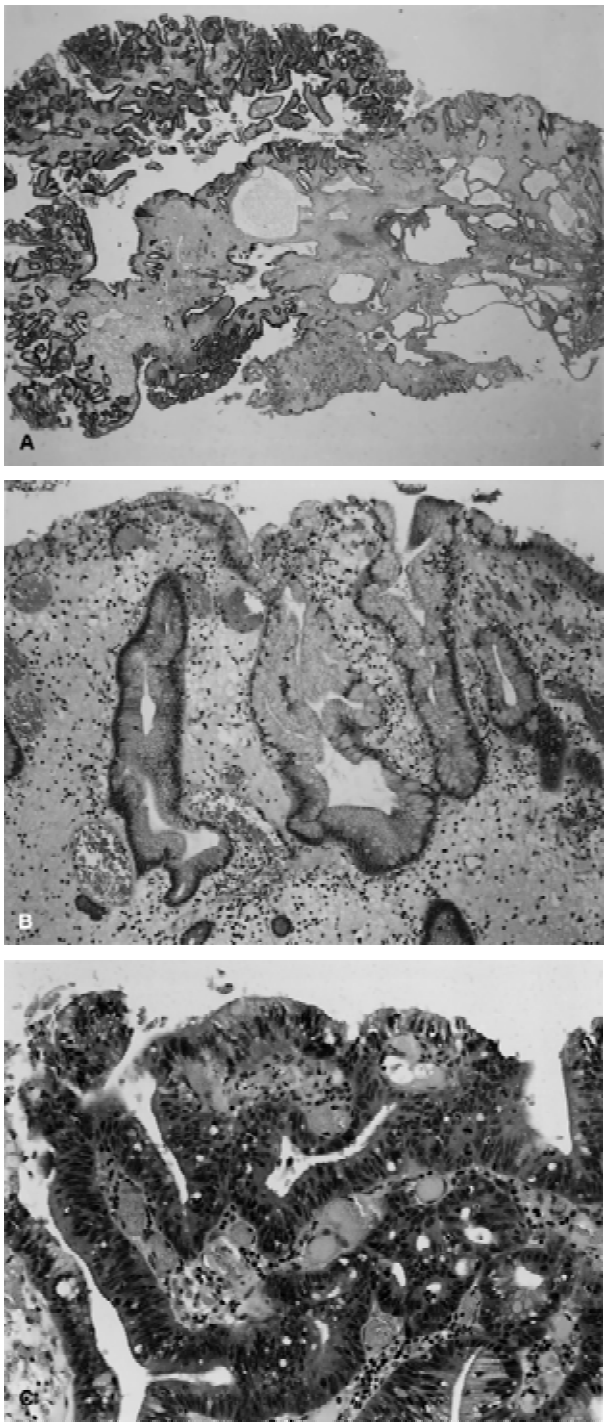

FIG. 1. Histologic features of a hyperplastic polyp. (**A**) At low magnification, the polyp is characterized by elongated, dilated foveolae and an edematous stroma. (**B**) The area of the polyp that is negative for dysplasia. Foveolae contain small, basally oriented nuclei and abundant cytoplasmic mucin. (**C**) The area of the polyp with high-grade epithelial dysplasia. Foveolae show marked nuclear stratification and hyperchromasia with an area of glandular cribriforming.

The following clinical, endoscopic, and histologic features were evaluated in each patient: age, gender, location of polyp(s) in the stomach, presence of single or multiple polyps, polyp size, presence of intestinal metaplasia in the polyp(s) and in the nonpolypoid gastric mucosa, presence of epithelial dysplasia in the polyp(s) and in the nonpolypoid mucosa, presence of adenocarcinoma in the polyp(s) and in the nonpolypoid mucosa, and the presence and histologic classification of gastritis in the surrounding gastric mucosa. Endoscopic biopsy sampling of the nonpolypoid gastric mucosa was also recorded (antrum only, body or fundus only, or both antrum and body or fundus) after or within 2 years of the index endoscopic examination.

Cases with intestinal metaplasia involving 25% or more of the biopsy tissue were classified as having extensive intestinal metaplasia; cases involving less than 25% of the biopsy tissue were classified as having focal intestinal metaplasia. Low-grade epithelial dysplasia was recorded based on the presence of nuclear enlargement, hyperchromatism, and stratification.[12,33] Cases with these nuclear features in a background of marked active inflammation or erosion or with milder nuclear hyperchromatism and stratification were graded as indefinite for dysplasia. Cases with severe cytologic atypia, glandular cribriforming, or full-thickness nuclear stratification were recorded as high-grade dysplasia (Fig. 1).[12,33]

Mucosal pathology in the background stomach was classified as follows: metaplastic atrophic gastritis of the autoimmune type (autoimmune gastritis) (corpus predominant); metaplastic atrophic gastritis of the environmental type (antral predominant); chemical or reactive gastropathy; reactive gastropathy in the postantrectomy or postgastroenterostomy stomach; gastric antral vascular ectasia (watermelon stomach); cytomegalovirus gastritis; peptic ulcer disease in the setting of Zollinger-Ellison syndrome; *H. pylori* gastritis; active chronic superficial gastritis suspicious for *H. pylori* but without organisms identified on Diff-Quik or *H. pylori* immunostains; lymphocytic gastritis; and normal or only mild chronic superficial gastritis. We defined autoimmune gastritis histologically as gastric body- and fundus-predominant glandular atrophy with intestinal, antral, or pancreatic acinar metaplasia and linear or nodular neuroendocrine cell hyperplasia on chromogranin immunostains.[35] Metaplastic atrophic gastritis of the environmental type was defined as replacement of a minimum of 25% of the antral surface and glandular epithelium by intestinal metaplasia on biopsy or resection material.[8,42] The histologic features of chemical or reactive gastropathy (i.e., foveolar hyperplasia, fibromuscular replacement of the lamina propria, lamina propria edema, vascular ectasia, and mucous depletion of the surface epithelium) were not formally quantitated; only cases with prominent changes were classified as chemical or reactive gastropathy.[14] Lymphocytic gastritis was defined as the presence of a minimum of 25 lymphocytes per 100 surface epithelial cells.[8,41]

Statistical Analysis

χ^2 analysis was used to compare the distribution of hyperplastic polyps and to compare the likelihood of multiple hyperplastic polyps between patients with and without autoimmune gastritis. A two-tailed p value of less than 0.05 was considered statistically significant.

RESULTS

Clinical and Endoscopic Findings

There was a slight female predominance, with 93 women (58%) and 66 men (42%). In one consultation case, the patient's gender was not recorded. Hyperplastic polyps were not seen in pediatric patients (younger than 18 years); the average patient age was 65.5 years (range, 22–88 years). The indications for endoscopy were given in 103 patients and included gastrointestinal bleeding (40 patients), abdominal pain or dyspepsia (27 patients), surveillance (e.g., Barrett esophagus, peptic ulcer disease, polyps or carcinoid tumors [22 patients]); heartburn, dysphagia, or esophagitis (17 patients); nausea or vomiting (9 patients); weight loss (4 patients); diarrhea (3 patients); evaluation for esophageal varices (1 patient); and evaluation of abdominal mass (1 patient). Sixteen patients had more than one indication for endoscopy.

On endoscopic examination, hyperplastic polyps were multiple in 37 (23%) patients (during the 1996–2000 study period in 32 patients and before this period in an additional five patients) and single in 128 (80%) patients. The antrum was the most common location of hyperplastic polyps, which were distributed as follows: antrum only in 96 (60%) patients, body or fundus only in 46 (29%) patients, cardia only in four (2.5%) patients, and antrum and body in 10 (6%) patients. In the remaining four (2.5%) patients, the location of the polyp was not stated. Most hyperplastic polyps were small (72% of the recorded sizes were less than 1 cm) and ranged from 0.2 to 9 cm. A summary of the clinical and endoscopic findings in patients with hyperplastic polyps is shown in Table 1.

Histologic Findings in Hyperplastic Polyps

Focal intestinal metaplasia was present in one or more hyperplastic polyps in 26 (16%) patients. Most hyperplastic polyps were not accompanied by epithelial dysplasia. Only three (2%) patients had high-grade dysplasia in the polyp (one was a mixed tubulovillous adenoma and hyperplastic polyp). Three (2%) patients had low-

TABLE 1. *Clinical and endoscopic findings in patients with hyperplastic polyps*

	No. (%)* of patients
Gender distribution	
Male	66 (42)
Female	93 (58)
Age (yrs) [mean (range)]	65.5 (22–88)
Polyp location	
Antrum only	96 (60)
Body/fundus only	46 (29)
Cardia only	4 (2.5)
Antrum and body	10 (6)
No. of hyperplastic polyps	
Single	128 (77)
Multiple	37 (23)
Polyp size† (cm)	
<0.5	58 (47)
0.5–1	31 (25)
1–2	22 (18)
2–3	8 (6)
>3	5 (4)

* Of 160 patients; however, not all findings were available for each patient.

† In patients with more than one hyperplastic polyp, size was based on the largest polyp. Cases described endoscopically only as a "nodule" or "small polyp" were classified as <0.5 cm. In 34 patients the size of the polyp was not recorded.

grade dysplasia in the polyp, and four (2.5%) patients had epithelial changes indefinite for dysplasia in the hyperplastic polyp. Carcinoma in the hyperplastic polyp was present in only one (0.6%) patient, a poorly differentiated carcinoma of the signet ring cell type that was present in multiple foci involving the polyp and the nonpolypoid stomach. It was therefore not possible to ascertain the exact site of origin of the carcinoma. Histologic findings in the hyperplastic polyps are summarized in Table 2.

Histologic Findings in the Surrounding Gastric Mucosa

Nine (6%) patients had synchronous or metachronous gastric adenocarcinomas, including two patients with gastric adenocarcinoma resected before the diagnosis of hyperplastic polyp, three patients with synchronous adenocarcinoma of the cardia–gastroesophageal junction, and four patients with synchronous adenocarcinomas of the antrum or body. Synchronous or metachronous carcinoid tumors were present in six (4%) additional patients with autoimmune gastritis. Separate adenomatous polyps were present in two (1.2%) patients.

Evaluation of the background nonpolypoid gastric mucosa could not be performed in 23 patients for whom no biopsies of nonpolypoid mucosa were performed. Thirteen additional patients had biopsy specimens of nonpolypoid mucosa taken only from the body; 26 had biopsy specimens taken only from the antrum; and 98 had biopsy specimens taken from the body and antrum. Of the 137 patients who had at least one biopsy specimen taken from nonpolypoid mucosa, 50 (37%) had intestinal metaplasia in the surrounding gastric mucosa; the intestinal metaplasia was focal in 20 patients and extensive in 30 patients. Low-grade epithelial dysplasia in the metaplastic epithelium was present in one (0.7%) patient.

Classification of background gastritis in the 137 patients with biopsies of nonpolypoid mucosa showed that *H. pylori* gastritis, chemical or reactive gastropathy, and autoimmune gastritis were the most frequent associations with hyperplastic polyps. Thirty-four (25%) had active chronic *H. pylori* gastritis (six with accompanying erosions or ulcer site); 29 (21%) had chemical or reactive gastropathy (six with erosions or ulcer); and 16 (12%) had autoimmune gastritis (Figs. 2–4). Other associations included active chronic gastritis suspicious for *H. pylori* but without demonstrable organisms on Diff-Quik or immunostains, metaplastic atrophic gastritis of the environmental type, reactive gastropathy of the type seen after partial gastrectomy (Billroth I or II anastomosis or cardiac–jejunal anastomosis) (Fig. 5), lymphocytic gastritis, metaplastic atrophic gastritis of uncertain type, cytomegalovirus gastritis, gastric antral vascular ectasia, gastric amyloidopathy with patchy active gastritis, and Zollinger-Ellison syndrome. Some patients had more than one type of disease and were included in more than one category. Histologic findings in the surrounding gastric mucosa are summarized in Table 3.

Interestingly, among the 16 patients with autoimmune gastritis, hyperplastic polyps tended to be more proximal in distribution than in patients without autoimmune gastritis. In this group, polyps were almost equally divided in location between the antrum and body, with polyps only in the body or fundus in seven patients, only in the antrum in six patients, and in both locations in the remaining three patients ($p = 0.03$). Hyperplastic polyps were multiple in eight patients with autoimmune gastritis and single in the other eight patients. Patients with autoimmune gastritis were therefore more likely (50%) than the other patients (29 of 144, 20%) to have multiple polyps ($p = 0.01$).

TABLE 2. *Histologic findings within hyperplastic polyps*

	No. (%) of patients
Intestinal metaplasia	26 (16)*
Epithelial dysplasia	
Negative	150 (94)
Indefinite	4 (2.5)
Low-grade	3 (2)
High-grade	3 (2)
Adenocarcinoma	1 (0.6)†

* Intestinal metaplasia was focal within all 26 hyperplastic polyps.

† A poorly differentiated adenocarcinoma of signet ring cell type that was present both within the hyperplastic polyp and in multiple foci throughout the surrounding stomach.

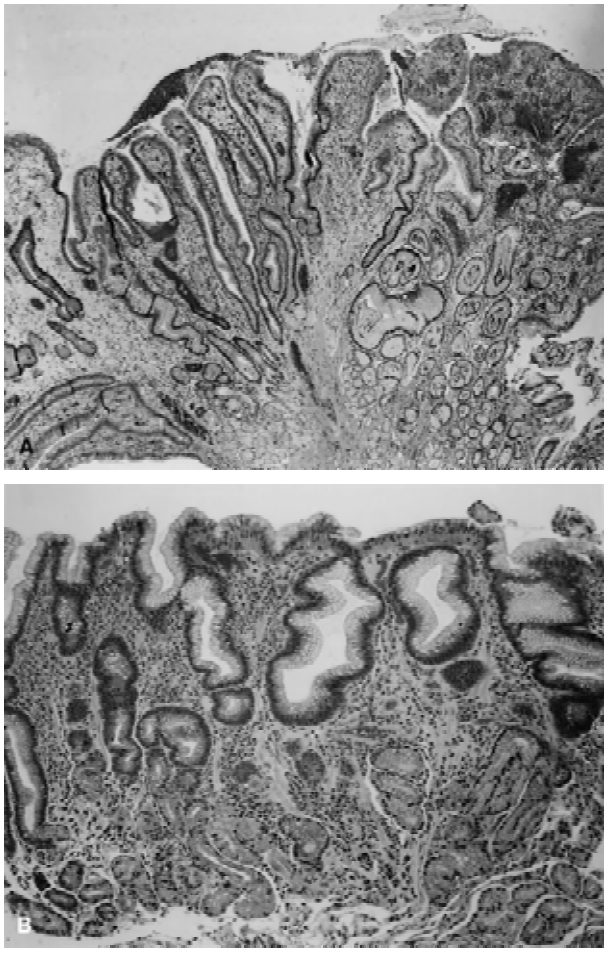

FIG. 2. Hyperplastic polyp from the antrum (**A**) in a patient with a superficial active chronic *H. pylori* gastritis in the nonpolypoid antral biopsy (**B**).

Only 21 (15%) patients had normal gastric biopsies or nonspecific histologic features, including mild chronic superficial inflammation or mild foveolar hyperplasia. Of these 21 patients, one had antral erosions endoscopically; one had a recent history of peptic ulcer disease; one had episodes of protracted vomiting; one was receiving alendronate (Fosamax [Merck, Whitehouse Station, NJ], an agent with the potential for caustic injury to the gastric and esophageal mucosa); and one had hypergastrinemia and clinical features suggestive of early autoimmune gastritis. In addition, 11 of the 21 patients had biopsy specimens taken from only the nonpolypoid antrum or body, but not both, suggesting that the true rate of normal or mild nonspecific mucosal disease is lower than 15%.

DISCUSSION

We have shown a high rate (85%) of background mucosal disease in the stomachs of patients with hyperplastic polyps. Particularly strong is the association between hyperplastic polyps and forms of chronic gastritis leading to mucosal atrophy and intestinal metaplasia. More than one third of patients had at least focal intestinal metaplasia in biopsy specimens of nonpolypoid mucosa, and more than one fifth had extensive intestinal metaplasia and glandular atrophy of autoimmune or environmental types. The findings in this study therefore confirm histologically many of the clinical observations of high rates of environmental (e.g., *H. pylori*) gastritis or serologic evidence of autoimmune gastritis (hypergastrinemia and antibodies to intrinsic factor and parietal cells) in patients with hyperplastic polyps.[16,22,23,25,27,28,39,40]

In addition to these high rates of *H. pylori* infection and autoimmune gastritis, other causes of gastric mucosal injury predisposing to hyperplastic polyp formation have been described in the literature, mainly in the form of case reports or small series,[5,9,11,20,36,38] but have not been previously evaluated in a comprehensive manner. We have shown hyperplastic polyps arising in association with such diverse forms of gastric disease as cytomegalovirus gastritis, gastric antral vascular ectasia, amyloidopathy, Zollinger-Ellison syndrome, and the postantrectomy stomach. In particular, the frequent association between chemical or reactive gastropathy and hyperplastic polyps has not previously been described. Chemical or reactive gastropathy, accompanied in many patients by erosions or ulcers, was second only to active *H. pylori* gastritis as the most common mucosal abnormality seen in the gastric mucosa other than the hyperplastic polyps in our patients (21% and 25% of patients, respectively). These findings underscore the important role of mucosal injury in the genesis of hyperplastic polyps and support the view of hyperplastic polyps as regenerative lesions.[26]

The association between hyperplastic polyps and autoimmune gastritis is of particular interest. These patients, in addition to comprising one of the most common groups with hyperplastic polyps, had a tendency to form multiple synchronous or metachronous polyps as compared with other patients ($p = 0.01$). Whether this is related to an intrinsic propensity for autoimmune gastritis to result in multiple hyperplastic polyps or simply reflects an artifact of increased endoscopic surveillance of these patients is not clear. Hyperplastic polyps in patients with autoimmune gastritis also showed a more proximal distribution in the stomach as compared with those in patients without autoimmune gastritis ($p = 0.03$).

Interestingly, despite the increased frequency of gastric body and fundus polyps in this group of patients, hyperplastic polyps were still found solely in the antrum in 6 (38%) of 16 patients with autoimmune gastritis. The reason for this antral distribution of hyperplastic polyps in a disease affecting predominantly the gastric body is

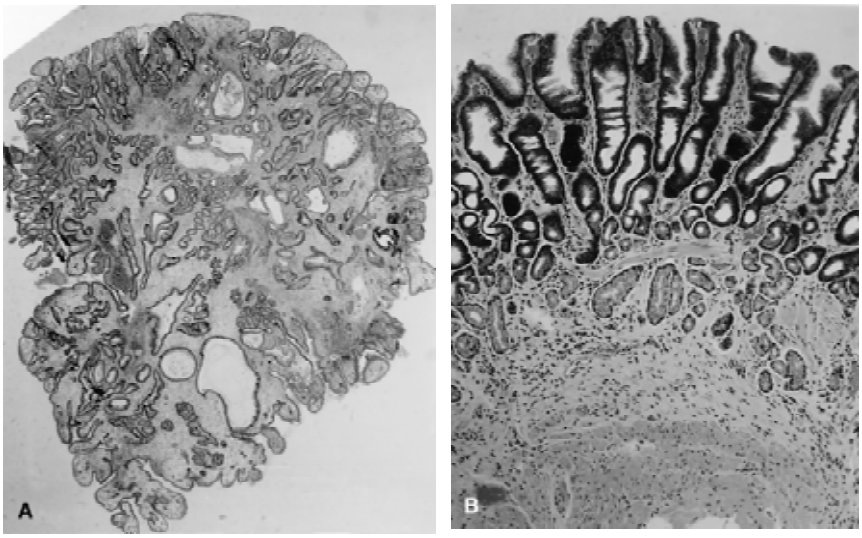

FIG. 3. Hyperplastic polyp from the antrum (**A**) in a patient with a nonpolypoid antral biopsy (**B**) showing marked foveolar hyperplasia, depletion of cytoplasmic mucin, smooth muscle stranding in the lamina propria, and mild superficial vascular ectasia, characteristic of chemical or reactive gastropathy.

not known. We have frequently noted the presence of varying degrees of foveolar hyperplasia in the antrum of patients with autoimmune gastritis (unpublished observations). The possible relationship between the histologic findings of foveolar hyperplasia in these patients, in some cases accompanied by fibromuscular proliferation in the lamina propria, and more characteristic cases of chemical or reactive gastropathy as seen in nonatrophic stomachs is unclear; we did not include these patients in the category of chemical gastropathy in this study. Laxen et al.[23] have also noted the frequent presence of antral hyperplastic polyps in patients with body-predominant (type A) atrophic gastritis and hypothesized a trophic effect of hypergastrinemia and duodenal reflux on the antral mucosa in these patients. Regardless of the exact cause of hyperplastic polyps in patients with autoimmune gastritis, it is important to recognize that an antral or even pyloric distribution of hyperplastic polyps in no way mitigates the possibility of significant disease in the gastric body.

The pathogenesis of hyperplastic polyps at the molecular level is unknown. Dijkhuizen et al.[7] have shown the presence of identical mutations in codon 12 of the K-*ras* oncogene in several concurrent hyperplastic polyps from the same patient, suggesting a clonal origin and malignant potential of these lesions. All the polyps in that patient had high-grade epithelial dysplasia, however, and one had an intramucosal adenocarcinoma. Other series of patients with hyperplastic polyps have reported much lower rates of epithelial dysplasia, ranging from 1.9% to 19%.[4,5,10,18,19] In this study, we found low- or high-grade dysplasia in only 4% of patients with hyperplastic polyps, using the criteria of Goldstein and Lewin[12] and Rugge et al.[33]

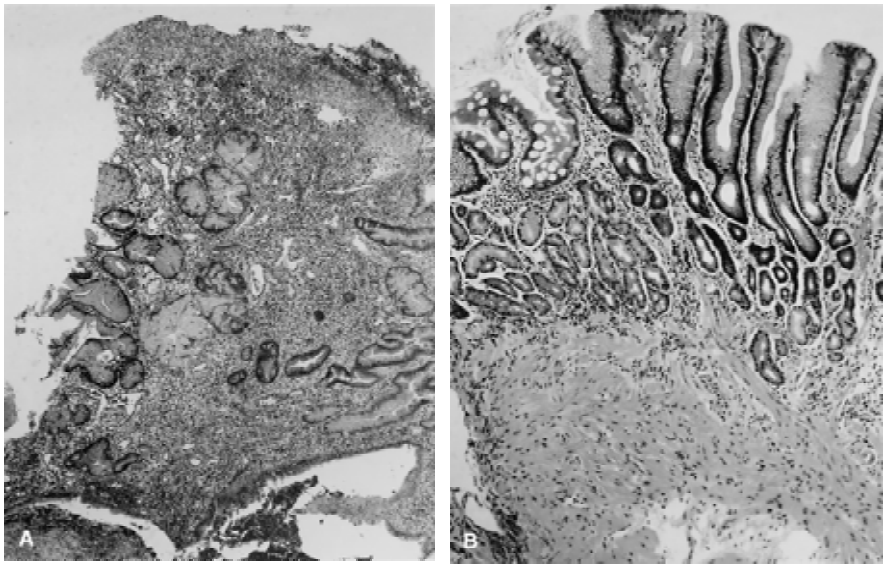

FIG. 4. (**A**) Hyperplastic polyp from the body in a patient with autoimmune gastritis. (**B**) Biopsy from the nonpolypoid body shows absence of parietal cells, intestinal and pyloric metaplasia, and a chronic inflammatory infiltrate in the lamina propria.

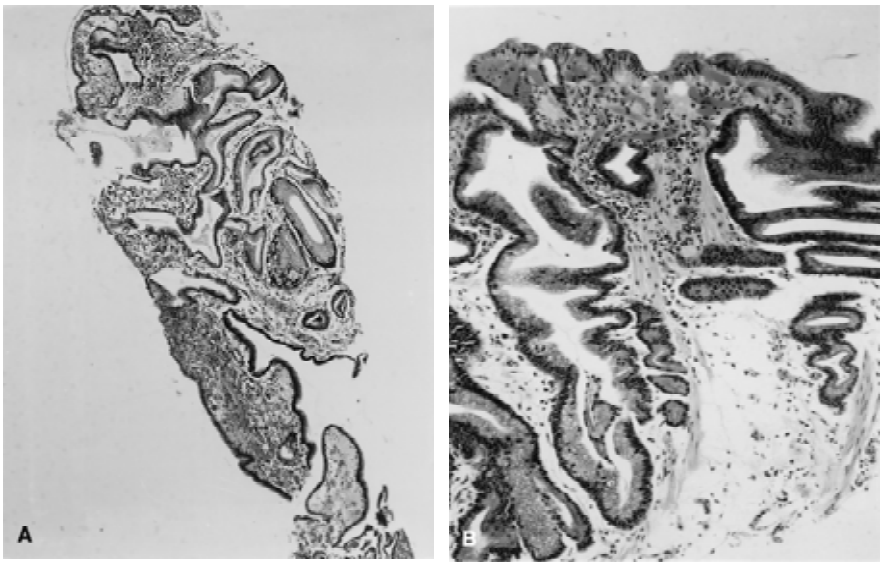

FIG. 5. (**A**) A hyperplastic polyp from the gastric remnant in a patient who had Billroth II gastrectomy. (**B**) Biopsy of the nonpolypoid mucosa near the anastomotic site shows characteristic atrophy of parietal cells, severe foveolar hyperplasia, and lamina propria edema.

Similarly, the frequency of infiltrating adenocarcinoma arising in hyperplastic polyps has been debated, with reported rates ranging from 0% to 13.5%.[3,4,6,10,15,18,21,28,31,37,44] In this series of 160 patients, only one had adenocarcinoma in a hyperplastic polyp, which represented a multifocal signet ring cell carcinoma that was also present in multiple areas of the stomach outside the polyp. Therefore, although unequivocal cases of adenocarcinoma arising in hyperplastic polyps have been described,[2,7,11,13,29,30,32,43] our data support that malignant transformation of hyperplastic polyps is a rare occurrence and that hyperplastic polyps are in general not premalignant lesions. The gastric mucosa outside the hyperplastic polyp is more likely than the polyp itself to harbor an adenocarcinoma (6% vs 0.6% in this series).

TABLE 3. *Histologic findings in surrounding gastric mucosa*

	No. (%) of patients
Intestinal metaplasia*	50 (37)
Focal	20 (15)
Extensive	30 (22)
Adenoma/flat dysplasia*	3 (2)
Adenocarcinoma	9 (6)
Carcinoid tumor	6 (4)
Background gastritis*†	116 (85)
H. pylori gastritis	34 (25)
Chemical/reactive gastropathy	29 (21)
Metaplastic atrophic gastritis	
Autoimmune type (corpus predominant)	16 (12)
Environmental type (antral predominant)	11 (8)
Uncertain type	3 (2)
Suspicious for *H. pylori*‡	14 (10)
Postgastrectomy	5 (4)
Lymphocytic gastritis	4 (3)
CMV gastritis	3 (2)
Gastric antral vascular ectasia	2 (1.5)
Zollinger–Ellison syndrome§	2 (1.5)
Amyloidopathy	1 (0.7)

CMV, cytomegalovirus.

* Based on 137 patients who had biopsies from at least one site in the nonpolypoid gastric mucosa.

† Some patients had more than one type of mucosal pathology and were included in more than one category.

‡ Active chronic superficial gastritis without organisms identified on Diff-Quik/*H. pylori* immunostains.

§ One patient with confirmed Zollinger–Ellison syndrome and one patient with hypergastrinemia, gastric carcinoid tumor, and nodular ECL cell hyperplasia suspicious for Zollinger–Ellison syndrome.

The true importance of hyperplastic polyps therefore appears to lie most significantly in their status as markers for an abnormal gastric mucosal background than as isolated preneoplastic lesions. Among the 137 patients in this study who had at least one biopsy for evaluation of nonpolypoid gastric mucosa, only 21 (15%) patients had normal or only nonspecific findings, including superficial chronic inflammation. The true rate of a normal or only mildly abnormal gastric mucosal background in patients with hyperplastic polyps is probably lower than 15%, based on two lines of evidence. First, 5 of these 21 patients had endoscopic or clinical features suggestive of gastric disease, including history of endoscopic erosions or ulcer, history of severe protracted vomiting, history of ingestion of caustic agents (alendronate), and hypergastrinemia. Second, 11 of the 21 patients did not have what we considered to be the minimum biopsy evaluation of nonpolypoid mucosa (i.e., biopsy specimens from the nonpolypoid antrum and body).

These findings underscore an unfortunate but not uncommon tendency among endoscopists for incomplete biopsy sampling, including in some cases, biopsy specimens taken only from the endoscopically visible lesions (as in 14% of our patients). Unlike other common gastric polyps, such as fundic gland polyps, which have little association with background gastric disease,[16,24,34] hyperplastic polyps arise most commonly in a background

of abnormal and often atrophic gastric mucosa. Pathologists diagnosing hyperplastic polyps in patients who have not had adequate biopsies of the nonpolypoid stomach should reiterate this association and recommend that biopsies of the surrounding mucosa be taken for evaluation of underlying gastric abnormalities. □

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