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【 ORIGINAL ARTICLE 】

Mucosal Atrophy of the Fornix as an Important Endoscopic Finding for an Autoimmune Gastritis Diagnosis

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Abstract:

Objective This study investigated the significance of diagnosing an endoscopic atrophic pattern of the fornix in autoimmune gastritis (AIG).

Materials Of 10,608 individuals (men/women: 6,551/4,057) who underwent an esophagogastroduodenal endoscopy (EGD) examination between April 2016 and March 2022 for a medical checkup, 80 patients (men/women: 34/46, mean age 58.7 years old) were diagnosed with AIG based on endoscopic findings and positivity for gastric autoantibodies. The mucosal atrophy pattern of the fornix shown in the endoscopic findings of AIG cases was divided into four classifications: none, patchy, wide-range, and whole.

Results The number of AIG cases classified as none, patchy, wide-range, and whole was 9, 26, 14, and 31, respectively. Whole-fornix atrophy was frequently observed in *Helicobacter pylori*-uninfected cases, while the atrophic pattern was correlated with the area of remnant oxyntic mucosa in the gastric body. The serum levels of pepsinogen I and pepsinogen I/II ratio decreased, while the gastrin level increased in association with the enlarged atrophic area of the fornix. The pepsinogen I/II ratio was ≥3.0, in 70.8% of the cases, with a patchy pattern. Follow-up EGD findings obtained after the AIG diagnosis showed gradual progression of mucosal atrophy of the fornix, with such progression primarily observed in *H. pylori*-eradicated cases.

Conclusion The degree of endoscopic atrophy of the fornix was correlated with AIG progression. Patchy atrophy pattern in the fornix may be helpful in the detection of AIG prior to oxyntic gland atrophy expansion.

Key words: autoimmune gastritis, endoscopy, fornix, atrophy, diagnosis

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Introduction

Autoimmune gastritis (AIG), also known as type A gastritis, is a chronic inflammatory disease of the stomach caused by cellular and humoral immune responses to the gastric parietal cells (1-3). Inflammation and continuous atrophy mainly occur in the gastric body of patients with AIG because parietal cells are epithelial cells located in the glands of the gastric corpus (2, 3). Recently, diagnostic criteria for AIG were established by the "Study Group on the establishment of diagnostic criteria for type A gastritis" of the Japan Gastroenterological Endoscopy Society (JGES) (4). Based on these criteria, the diagnosis of AIG is confirmed when endoscopic and/or histological findings meet the requirements for AIG, and the patient is positive for gastric autoantibodies (either anti-parietal cell, anti-intrinsic factor antibodies, or both).

Although several endoscopic findings of the gastric body, such as corpus-predominant advanced atrophy and sticky adherent dense mucus, have been reported in cases of AIG (4-6), we have observed different patterns, indicating varying degrees of endoscopic atrophy in the gastric fornix of affected patients. However, to our knowledge, the significance of endoscopic observation of mucosal atrophy of the fornix area in patients with AIG has not been reported.

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The present study was thus performed to investigate endoscopic atrophic patterns of the fornix and their usefulness for the estimation of AIG progression as well as detection of suspected cases.

Materials and Methods

The study subjects were selected from individuals who underwent esophagogastroduodenal endoscopy (EGD) as part of a detailed medical checkup at the Health Center of Shimane Environment and Health Public Corporation between April 2016 and March 2022. In our institute, it was not possible to determine the presence of serum gastric autoantibodies in the stored serum of individuals with suspected AIG based on endoscopic findings who were examined after April 2022 due to stoppage of outsourced measurements. During the study period, 29,204 EGD examinations were performed on 10,608 individuals (men/women: 6,551/4,057) without a history of gastrectomy. Each examination was conducted by an experienced endoscopist who had received JGES certification and/or more than five years of experience with EGD using an EG-L580NW endoscope (FUJIFILM, Tokyo, Japan). Endoscopic findings indicating suspected AIG were corpus-predominant advanced atrophy, sticky adherent dense mucus in the fornix and upper body, flat and elevated remnant oxyntic gland mucosa (ROM) in a diffuse atrophic area, and hyperplastic polyp (4-6). Gastric mucosal atrophy (GMA) extends from the antrum to the corpus in cases with *Helicobacter pylori* infection, and endoscopic findings are divided into six groups (C1, C2, C3, O1, O2, and O3), based on the classification of Kimura and Takemoto (7). This classification clearly demonstrates that GMA consecutively extends from the 1) antrum to the body, 2) lesser curvature of the lower body to the upper body, and 3) lesser curvature to the greater curvature of the body. However, irregular atrophic patterns not included in that classification, such as corpus-predominant and multiple partial mucosal atrophy sites in the fundic gland area, are frequently observed in cases with AIG; thus, such affected individuals were also diagnosed with suspected AIG.

Serum samples were obtained from all suspected AIG cases and stored at -80°C, then gastrin level, and concentrations of anti-parietal cells and anti-intrinsic factor antibody were determined by SRL (Tokyo, Japan). For the analysis of gastrin, a radioimmunoassay procedure with a polyethylene glycol method was used, while anti-parietal cells and anti-intrinsic factor antibodies were analyzed using a fluorescent antibody test and a chemiluminescent enzyme immunoassay method, respectively. A positive finding for anti-parietal cells was determined when the titer was ≥10 U. For the present study, a subject with endoscopic findings resulting in suspected AIG was diagnosed as having AIG when anti-parietal cells and/or anti-intrinsic factor antibodies were found to be positive. The serum levels of pepsinogen I and II were determined using SphereLight Pepsinogen I and II kits (FUJIFILM Wako Pure Chemical, Osaka, Japan), and the pepsinogen I/II ratio was calculated. Cases with pepsinogen I/II ≥3.0 were also investigated, as past studies have shown that a value below 3.0 indicates severe fundic gland atrophy (8, 9).

During the study period, 80 individuals were diagnosed with AIG based on endoscopic findings indicating suspected AIG and positive for gastric autoantibodies, and were enrolled as subjects. Of these, positive serum findings for anti-parietal cells were noted in 79, while anti-intrinsic factor antibodies were noted in 6, with 5 positive cases for both. There were 34 men and 46 women, and the mean age at the time of the AIG diagnosis was 58.7±10.5 (range: 35-81) years old. The prevalence of AIG was 0.75% among the 10,608 individuals during the study period, while the numbers of AIG cases in the 6,551 men and 4,057 women were 34 (0.52%) and 46 (1.13%), respectively ($p<0.001$).

A public health nurse conducted an interview with each subject to obtain a precise medical history. The presence or absence of coexisting autoimmune diseases, including autoimmune thyroid disease, was also investigated. *H. pylori* infection status was comprehensively determined as positive, post-eradication, or uninfected based on these findings, as well as diagnostic testing for that infection and EGD results. During the study period, the serum anti-*H. pylori* IgG antibody assays for determining *H. pylori* infection were performed using the SphereLight *H. pylori* antibody J kit or L-type Wako *H. pylori* antibody J kit (FUJIFILM Wako Pure Chemical) (10, 11). Patients who had undergone therapy without successful eradication were not included in the post-eradication group. When eradication therapy could not be confirmed as successful, an *H. pylori* stool antigen test at our hospital was recommended. Those without successful eradication were included in the *H. pylori* infection group, even if they had previously undergone eradication therapy. The presence or absence of *H. pylori* infection and successful eradication were confirmed based on endoscopic findings (12-14).

To investigate the significance of an atrophic pattern in the fornix, endoscopic findings were divided into four classifications: none, patchy, wide-range, and whole. Patchy atrophy was defined as mucosal atrophy that had spread to less than 50% of the fornix. Dotted pattern atrophy was observed in the patchy atrophy pattern. Wide-range atrophy was determined when mucosal atrophy had spread to more than 50% of the fornix area, and whole atrophy when spreading throughout the area of the fornix, and no ROM in an area of diffuse mucosal atrophy was observed. This endoscopic atrophic pattern classification was considered to indicate the width of mucosal atrophy in the fornix. The atrophic findings can be more easily determined by endoscopic observation using linked color imaging (LCI) or blue laser imaging (BLI) (Fig. 1, 2). However, some characteristic histological findings, such as protrusion into the lumen or degeneration of parietal cells, were not noted in endoscopically biopsied specimens obtained from the fornix in some cases diagnosed as AIG (data not shown).

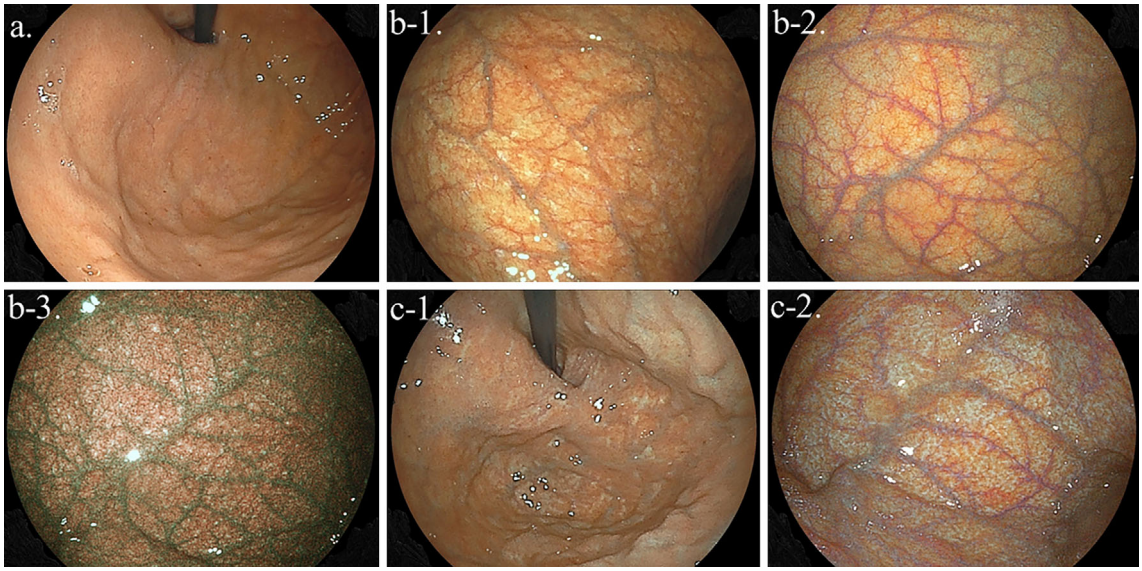

Figure 1. **Endoscopic images showing mucosal atrophy patterns of fornix. a: None. b: Patchy (dotted) pattern [b-1: white light imaging (WLI), b-2: linked color imaging (LCI), b-3: blue laser imaging (BLI)]. The dotted mucosal atrophic pattern was easily determined based on endoscopic observations with LCI or BLI compared to WLI. c: Patchy pattern (c-1: WLI, c-2: LCI). Patchy mucosal atrophic patterns were easily determined based on endoscopic observations with LCI compared with WLI.**

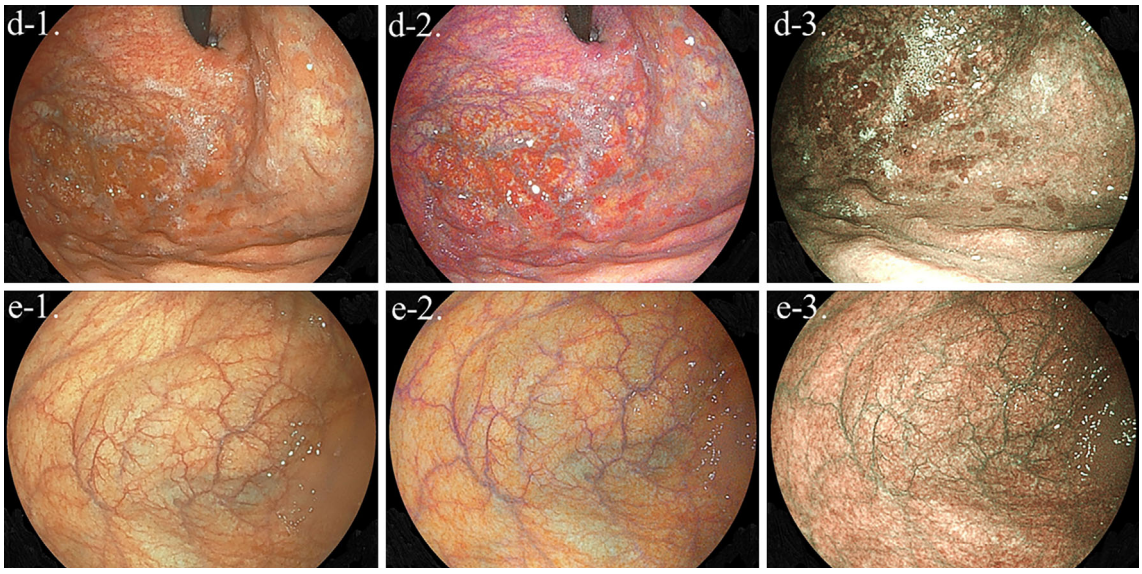

Figure 2. **Endoscopic images showing mucosal atrophy patterns of fornix. d: Wide-range pattern [d-1: white light imaging (WLI), d-2: linked color imaging (LCI), d-3: blue laser imaging (BLI)]. The remnant oxyntic mucosa was easily determined based on endoscopic observations with LCI and BLI compared to WLI. e: Whole pattern of mucosal atrophy (e-1: WLI, e-2: LCI, e-3: BLI). Remnant oxyntic mucosa was not observed in the fornix.**

Recently, endoscopic classification of ROM areas in the gastric body or fornix in AIG cases was reported by Maruyama et al., with division into early-stage 1) for >50% to 100%, mid-stage 2) for >10% to 50%, and end-stage 3) for ≤10%, reflecting disease progression (15). In the present study, that classification was used, with the area of ROM in the gastric body receiving focus for a comparison with the degree of fornix mucosal atrophy (Fig. 3). Comparisons of serum levels of gastrin and pepsinogen I and II and the pepsinogen I/II ratio among the different degrees of mucosal atrophy of the fornix were also performed. In addition, the fornix atrophy pattern in patients with AIG was examined during a follow-up EGD examination after diagnosis to determine possible atrophy progression.

All endoscopic images from each subject were simultaneously reviewed by three expert endoscopists to determine

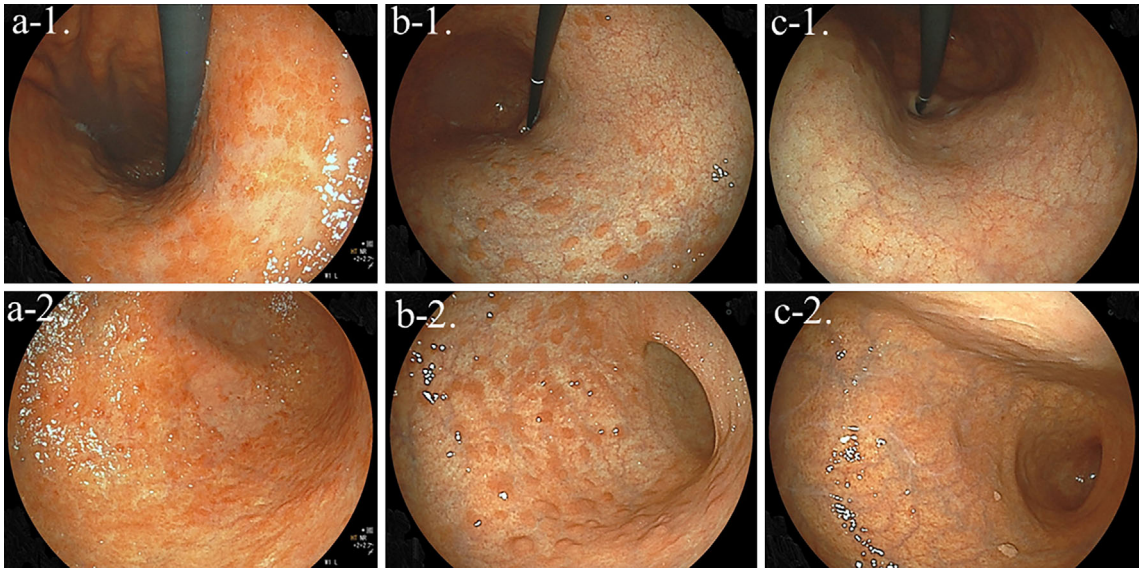

Figure 3. Classification of remnant oxyntic mucosa (ROM) area in gastric body. a: >50% to 100%, b: >10% to ≥50%, c: ≤10% (a-1, b-1, c-1: lesser curvature of gastric body, a-2, b-2, c-2: greater curvature of gastric body).

the degree of GMA, findings related to *H. pylori* infection, suspected AIG, fornix mucosal atrophic pattern, and ROM classification in the gastric body with decisions made by consensus. When there was inconsistency in judgment concerning the endoscopic images among the experts, the final diagnosis was decided by the lead endoscopist (K.A.).

Statistical analyses were performed using the chi-square test, Fisher's exact test, Kruskal-Wallis test, and Mann-Whitney U test. All calculations were performed using the Stat View 5.0 software program (Abacus Concepts, Berkeley, USA) for Macintosh, with a p value <0.05 considered to indicate statistical significance.

This study was performed in accordance with the Declaration of Helsinki, and the protocol was approved by the Ethics Committee of Shimane Environment and Health Public Corporation (No. 201703-202305). Written informed consent indicating that clinical data would be used for a clinical study without the release of individual information was obtained from all subjects before performing the medical checkup examinations.

Results

Subject characteristics, findings indicating the relationship between the fornix atrophic pattern and ROM area in the gastric body, and serum pepsinogen and gastrin levels are presented in Table 1. Regarding the atrophic pattern in the fornix, the numbers of cases with none, patchy, wide-range, and whole were 9, 26, 14, and 31, respectively. Age gradually increased in association with progression of mucosal atrophy in the fornix, although no significant differences were observed. An analysis of the relationship between the endoscopic atrophic pattern of the fornix and *H. pylori* infection status showed whole atrophy of the fornix at the time of the AIG diagnosis in 18 of the 31 *H. pylori*-uninfected cases. The number of AIG cases with autoimmune thyroid disease (Hashimoto's or Basedow's thyroiditis) was 14 (17.5%), and autoimmune thyroid disease was more frequent in patients with a higher degree of mucosal atrophy of the fornix than in others. Autoimmune diseases other than thyroid disease were not observed in our patients with AIG.

The serum pepsinogen I level was significantly different among the different atrophic patterns, with a decrease associated with the progression of endoscopic mucosal atrophy in the fornix. Furthermore, the pepsinogen I/II ratio tended to decrease, and serum gastrin levels tended to increase with the progression of mucosal atrophy of the fornix. The mean pepsinogen I/II ratio for cases categorized as none and patchy atrophy pattern was 3.9 and 3.5, respectively, while the number of cases with a pepsinogen I/II ratio ≥3.0 in those groups was 6 (75.0%) and 17 (70.8%), respectively.

The fornix atrophic pattern in AIG cases was correlated with the gastric body ROM area. In the 25 cases with stage 1 ROM, those with none, patchy, wide-range, and whole atrophic patterns numbered 8, 12, 2, and 3, respectively. Thus, 20 (80%) of the cases with stage 1 were classified as none and patchy atrophic pattern, while 5 (20%) had a wide-range or whole atrophic pattern. As for the 55 cases with stage 2 or 3 ROM in the gastric body, none, patchy, wide-range, and whole atrophic pattern was observed in 1, 14, 12, and 28 cases, respectively. Among these cases, 40 (73%) showed a wide-range or whole atrophic pattern, and 15 (27%) showed a none or patchy atrophic pattern.

A follow-up EGD examination after confirmation of the AIG diagnosis was performed in 47 of the 80 subjects. During the mean follow-up period of 4.6 years, no progression of endoscopic mucosal atrophy of the fornix was observed in 38 (81%) of the examined cases. While progression of

Table 1. Characteristics of Auto-immune Gastritis Subjects with Different Endoscopic Atrophic Pattern of Fornix.

	Atrophic pattern of fornix				p value
	None	Patchy	Wide-range	Whole	
Number of subjects	9	26	14	31	
Male/female	3/6	14/12	5/9	12/19	0.547
Age, years, mean±SD	54.1±12.3	57.8±10.7	59.9±9.6	60.1±10.3	0.675
Status of *H. pylori* infection					0.047
Positive	2	2	2	3	
Eradicated*	3	19	8	10	
Uninfected*	4	5	4	18	
Autoimmune thyroid diseases	0	1	2	11	0.006
Hashimoto's thyroiditis	0	1	2	7	
Basedow's thyroiditis	0	0	0	4	
Pepsinogen I (ng/mL), mean±SE**	44.5±15.5	32.2±3.9	33.6±8.8	15.9±2.4	0.005
Pepsinogen II (ng/mL), mean±SE**	11.1±3.3	9.9±1.2	14.9±4.3	8.8±1.2	0.359
Pepsinogen I/II ratio, mean±SE**	3.9±0.7	3.5±0.3	2.6±0.5	2.3±0.4	0.037
Pepsinogen I/II ratio ≥3.0**	6 (75.0%)	17 (70.8%)	5 (38.5%)	10 (34.5%)	0.022
Serum gastrin (pg/mL), mean±SE**	210±98	523±206	878±250	1,067±240	0.037
Remnant oxyntic mucosa***					p<0.001
Stage 1 (>50%)	8 (88.9%)	12 (46.2%)	2 (14.3%)	3 (9.7%)	
Stage 2 (>10% to 50%)	0	11 (42.3%)	7 (50.0%)	7 (22.6%)	
Stage 3 (≤10%)	1 (11.1%)	3 (11.5%)	5 (35.7%)	21 (67.7%)	

*Significant difference between *H. pylori*-eradicated and uninfected subjects.

**Pepsinogen I, II, and I/II ratio, and serum gastrin calculated after excluding six subjects with anti-secretory drug administration.

***Remnant oxyntic mucosa (ROM) of gastric body.

mucosal atrophy was not observed in nearly all *H. pylori*-uninfected cases, 7 (25.9%) of the 27 *H. pylori*-eradicated cases showed slight progression (Table 2). EGD images from a representative post-eradication case of AIG showing progression of mucosal atrophy of the fornix over a five-year period are presented in Fig. 4. There were no significant differences in sex, age, coexisting autoimmune thyroid disease, duration after *H. pylori* eradication, or follow-up period after the AIG diagnosis between *H. pylori*-eradicated cases with and without progression of mucosal atrophy of the fornix (Table 3).

Discussion

AIG is caused by cellular and humoral immune responses to gastric parietal cells and leads to the development of chronic continuous inflammation and GMA in the body and fornix of the stomach (1-3). Recently, diagnostic criteria for AIG were established by the "Study Group on the establishment of diagnostic criteria for type A gastritis" of the JGES (4), attracting the attention of endoscopists due to the presence of gastric tumors, such as neuroendocrine tumor and gastric cancer, in affected individuals (5, 6). Among the 10,608 subjects who underwent EGD between April 2016 and March 2022, AIG was noted in 0.75%. In addition, our previous study noted AIG in 0.49% of 6,739 individuals who underwent EGD during the 2-year period from May 2016 to April 2018 (16). The criteria for the diagnosis of AIG were the same for both studies, with the higher rate noted in the present findings considered to be due to an increased understanding of this disease condition. In addition, several reports of endoscopic results in an early stage of AIG have recently been presented (17-19). Thus, the prevalence of AIG may continue to increase in the future. Gender prevalence in our previous study showed that 0.40% of men and 0.65% of women affected, while the present study showed 0.52% and 1.13%, respectively. Therefore, predominance among women remained despite the increased rate of AIG diagnoses.

Characteristic endoscopic findings, including corpus predominant advanced atrophy, sticky adherent dense mucus in the fornix and upper body, flat and elevated ROM, and hyperplastic polyps, have been reported in cases of AIG (4-6). In addition, endoscopic findings of early-stage AIG, such as reddish nodules, longitudinal arrangement of pseudopolyps (bamboo joint-like appearance), and swelling of the gastric area with erythema (salmon roe-like appearance), have also been noted (17-19). However, these endoscopic findings at an early stage focused on mucosal changes in the gastric body. In contrast, endoscopic atrophic change of the fornix was noted in the present study, and its relationship with AIG progression was investigated. The results showed that the fornix atrophic pattern correlated with the ROM area in the gastric body. A patchy atrophic pattern of the fornix was primarily observed in patients with stage 1 ROM (>50% of the gastric body), while wide-range and whole atrophic pat-

Table 2. Relationship of Fornix Atrophic Pattern Shown by EGD at Time of AIG Diagnosis with Follow-up EGD Findings.

	Follow-up EGD			
	None	Patchy	Wide-range	Whole
EGD at time of diagnosis				
None	2	1		
Patchy		13	3	1
Wide-range			6	4
Whole				17
H. pylori-uninfected				
None	1			
Patchy		2	1	
Wide-range			2	
Whole				10
H. pylori-eradicated				
None	1	1		
Patchy		10	2	1
Wide-range			4	3
Whole				5
H. pylori-eradication*				
None				
Patchy		1		
Wide-range				
Whole				1
H. pylori-positive				
None				
Patchy				
Wide-range				1
Whole				1

Follow-up EDG was performed at 4.6±1.8 (1-8) years after EGD performed at time of diagnosis.

**H. pylori* eradication performed between EGD at time of diagnosis and follow-up EGD.

terns of the fornix were mainly observed in patients with stage 2 or 3 ROM (≤50%). Furthermore, there was a tendency for the serum level of pepsinogen I and I/II ratio to decrease, and the serum gastrin level increased by expansion of the atrophic area. Therefore, the endoscopic atrophy grade in the fornix is considered a valuable marker for estimating the progression of AIG.

In contrast, the present results showed that the degree of mucosal atrophy differed between the gastric body area and fornix in some subjects with AIG. In some cases with stage 1 ROM of the gastric body, a wide-range or whole atrophic pattern was noted, whereas no or patchy atrophic patterns were observed in some stage 2 and 3 cases. Endoscopic estimation of the ROM area in the gastric body is sometimes difficult in *H. pylori*-positive and *H. pylori*-eradicated cases because of mucosal atrophy induced by *H. pylori* infection as well as the large gastric body area. Conversely, patchy atrophic patterns of the fornix are considered to be easily assessed because of the smaller area of the fornix. Thus, attention to the atrophy pattern of the fornix during EGD might increase the detection of cases with suspected AIG. In more than two-thirds of the present cases with a patchy atrophy pattern, the pepsinogen I/II ratio was ≥3.0, and this atrophic pattern could be easily detected by endoscopic observations using LCI or BLI. Therefore, identification of the fornix atrophic pattern is thought to be helpful for an early-stage AIG diagnosis, although a large-scale prospective study will be needed to clarify the significance of endoscopic observations of the fornix for the diagnosis of suspected AIG cases.

The results of this study showed a higher degree of mucosal atrophy in *H. pylori*-uninfected AIG cases than in *H. pylori*-eradicated cases, and coexisting autoimmune thyroid diseases were frequently observed in cases with a higher degree of mucosal atrophy. Therefore, different mechanisms might be correlated with the occurrence of AIG between *H. pylori*-uninfected and *H. pylori*-eradicated cases, although we are unable to suggest possible underlying mechanisms at present.

Follow-up EGD after the AIG diagnosis revealed progression of endoscopic mucosal atrophy of the fornix in only 9 of 47 AIG cases (19%), although whole mucosal atrophy of the fornix was observed at the time of the AIG diagnosis in 17 cases (36%). Therefore, the progression of mucosal atrophy of the fornix in AIG is generally slow. In the present study, the time-course progression of atrophic areas in the fornix was mainly observed in *H. pylori*-eradicated AIG cases. We could not explain the factors related to the progression of mucosal atrophy of the fornix in *H. pylori*-eradicated cases because there was no significant difference in several investigated factors between those with and without progression of fornix mucosal atrophy. In our AIG cases, mucosal atrophy in the fornix area of *H. pylori*-eradicated cases was milder than that of *H. pylori*-uninfected cases. Therefore, *H. pylori*-eradicated AIG cases are considered to be diagnosed at an earlier stage of disease than *H. pylori*-uninfected cases. The follow-up period in our cases might be too short to determine the progression of mucosal atrophy in *H. pylori*-eradicated AIG cases, and a long-term prospective study is recommended to clarify the clinical course of these cases. Eradication of *H. pylori* infection has been reported to have an important effect on the occurrence of AIG (20-23), and some kind of altered immune response after *H. pylori* eradication is considered to be related to the occurrence and progression of AIG. Although the mechanisms underlying the occurrence and progression of AIG have not been clarified, a change in mucosal atrophy of the fornix is considered a valuable endoscopic finding for the detection of suspected AIG cases, especially in individuals with *H. pylori*-eradicated status.

Several limitations associated with the present study warrant mention. It was retrospectively performed at a single medical center, and the majority of the subjects were socially active and productive; thus, it included relatively few young or elderly individuals. Conversely, the subjects were considered representative of the general population com-

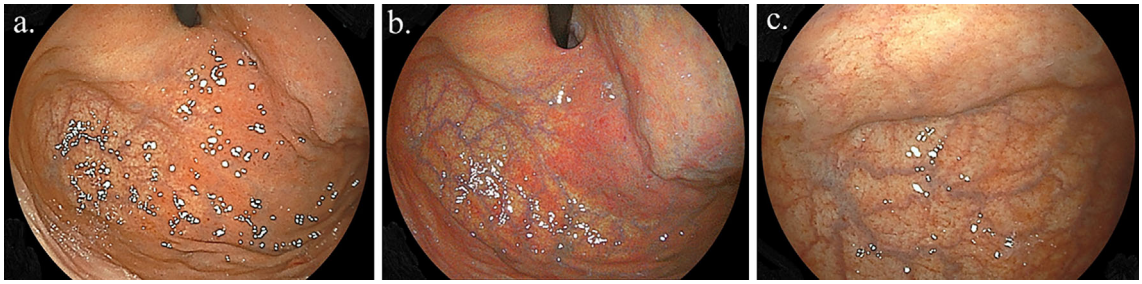

Figure 4. **Findings at the time of the AIG diagnosis of a 56-year-old woman who had undergone *H. pylori* eradication 2 years prior. The results indicated positivity for anti-parietal cells (titer 80-fold) and negative for anti-intrinsic factor antibody. The pepsinogen I/II ratio was 0.7 and level of gastrin in serum was 930 pg/mL. Endoscopic images at the time of the AIG diagnosis (a and b, b: linked color imaging) showed wide-range atrophy of the fornix, while whole mucosal atrophy was observed by endoscopic imaging performed five years later (c).**

Table 3. **Characteristics of *H. Pylori*-eradicated AIG Cases with and without the Progression of Mucosal Atrophy in Fornix Area.**

	Progression of mucosal atrophy in fornix		p value
	With	Without	
Number of cases	7	20	
Male/female	3/4	13/7	0.391
Age, years, mean±SD	61.1±4.9	60.6±11.0	0.868
Autoimmune thyroid diseases	1/6	3/17	0.964
Duration after *H. pylori* eradication*, mean±SD	4.7±2.4	5.1±2.0	0.740
Follow-up period**, mean±SD	5.0±1.5	4.3±1.8	0.361

*Duration between *H. pylori* eradication and the time of AIG diagnosis.

**Follow-up period after AIG diagnosis.

pared with patients who visited a medical clinic for specific symptoms and accompanying disease conditions. In this study, all endoscopic images were simultaneously reviewed by three expert endoscopists to determine several endoscopic findings. Therefore, inter-observer differences in endoscopic findings of the fornix could not be examined. Additional studies should be performed to determine the significance of endoscopic observation of the fornix for the detection of suspected AIG cases and to estimate disease progression in patients diagnosed with AIG.

In conclusion, the present results showed that the prevalence of AIG was 0.75% in Japanese individuals who underwent EGD as part of an annual checkup. The degree of atrophy of the fornix determined based on endoscopic findings correlates with AIG disease progression, while the presence of a patchy pattern may play an important role in the early detection of suspected cases.

The authors state that they have no Conflict of Interest (COI).

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