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Assigning a different endoscopist for each annual follow-up may contribute to improved gastric cancer detection rates

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ABSTRACT

Background and study aims Esophagogastroduodenoscopy (EGD) is an effective and important diagnostic tool to detect gastric cancer (GC). Although previous studies show that examiner, patient, and instrumental factors influence the detection of GC, we analyzed whether assigning a different examiner to surveillance EGD would improve the detection of GC compared to assigning the same examiner as in the previous endoscopy.

Patients and methods We retrospectively reviewed patients who underwent two or more consecutive surveillance EGDs at a single center between 2017 and 2019. We identified factors associated with GC detection using multivariable regression analysis and propensity-score matching.

Results Among 7794 patients, 99 GC lesions in 93 patients were detected by surveillance EGD (detection rate; 1.2 %), with a mean surveillance interval of 11.2 months. Among the detected 99 lesions, 87 (87.9 %) were curatively treated with endoscopy. There were no differences in the clinicopathologic characteristics of GC detected by the same or different endoscopists. GC detection in the group examined by different endoscopists was more statistically significant than in the group examined by the same endoscopist, even after propensity-score matching (1.6 % and 0.7 %; $P<0.05$). Endoscopic experience and other factors were not statistically significant between the two groups.

Conclusions In surveillance EGD, having a different endoscopist for each exam may improve GC detection rates, regardless of the endoscopist's experience.

Introduction

Gastric cancer (GC) is the fifth most common cancer and the third leading cause of cancer death worldwide [1]. Screening with esophagogastroduodenoscopy (EGD) leads to GC mortality reduction in a healthy population [2–4]. Previous studies have shown the usefulness of EGD in GC detection, and early GC treatment also reduces GC mortality (hazard ratio [HR] 0.51) [5, 6]. In addition, surveillance EGD after early GC treatment is important. High metachronous GC rates with a cumulative 3-year incidence of 5.6 % have been reported [7]. Although a Japanese cohort study of annual surveillance EGD reported that 96.2 % of metachronous GCs were curable with endoscopic resection [7], they can lead to death [8]. Therefore, long-term surveillance is important and an efficient endoscopic surveillance strategy is required.

To detect early GC with EGD and reduce GC mortality, it is necessary to improve the accuracy of EGD. Factors related to EGD accuracy include examiner, patient, and endoscopic instrument factors. There are many challenges in improving the quality of screening EGD. The rate of missed GC within 3 years of surveillance is high: 4.7 % as reported in a multicentric cohort

study [9], and 9.4% to 11.3% in a systematic review and meta-analysis [10, 11]. An endoscopist's experience can influence missed GC diagnoses. The false-negative rate of GC detection by EGD is approximately 11.3% to 25.8%, depending on the number of years of endoscopic experience: 32.4% for examiners with <10 years of experience and 19.5% for examiners with ≥10 years [12]. To prevent false negatives in GC detection, a systematic screening protocol for the stomach (SSS) [13], increasing the examination time [14], and increasing examiner experience [15, 16] are effective strategies.

The possibility of missing a diagnosis can be expected to an extent if the diagnosis is made by a lone physician. The collective intelligence of multiple physicians can improve diagnostic accuracy [17]. In colonoscopy, it was reported that adenoma detection rates are improved with nursing observation, or a "second set of eyes" [18]. Similarly, observation by multiple endoscopists may improve detection of GC with EGD, but the application methods and usefulness in surveillance EGD are not clear. We focused on the use of a "second set of eyes" per patient by assigning a different endoscopist for each follow-up examination. In this study, we analyzed examiner factors involved in GC detection with surveillance EGD and how the duration of experience and performance of the test by a same or different endoscopist each time affected the GC detection rate.

Patients and methods

Study cohort

We performed a retrospective cohort study at a single center (Sendai Kousei Hospital, Japan). Patients were included if they underwent two or more consecutive surveillance EGDs between March 2017 and October 2019. Patients with only one screening EGD, EGD for a closer examination of an already diagnosed cancer, or treatment EGD were excluded. To identify suitable cases, we reviewed endoscopy reports and electronic medical health records.

This study was conducted in accordance with STROBE guidelines and was approved by the Institutional Review Board of the Sendai Kousei Hospital (authorization number: 1–99). Informed consent was obtained comprehensively in an opt-out format.

Primary outcome

The primary outcome was GC detection, classified as detected or non-detected.

Variable measurements

We collected patient (age, sex, surveillance objective, *Helicobacter pylori* status) and endoscopy (sedative agent used, scope type, endoscopist, and time interval between EGD) characteristics. Mucosal atrophy was assessed endoscopically using the Kimura-Takemoto classification [19]. Information about mucosal atrophy was obtained from the initial endoscopy. The U portion included the cardia, fundus, and upper body; the M portion included the mid-body and lower body; and the L portion included the angle and antrum [20]. The macroscopic GC types were classified according to the Paris classification as type 0-I (protruded), type 0-IIa (superficially elevated), type 0-IIb (flat), type 0-IIc (superficially depressed), or type 0-IIa + IIc (elevated with a central depression) [21].

Surveillance EGD setting

The Endoscopic Department involved in the study site specializes in endoscopic diagnosis and treatment of patients with cancer and mainly receives referrals from local physicians. Most surveillance EGDs performed here are for cancer screening purposes and were targeted for those performed at approximately annual intervals. Endoscopists are assigned patients randomly on their assigned examination days. This accounts for the variability of the endoscopist for each surveillance EGD of the same patient. Twenty-nine endoscopists were engaged during the study period. The endoscopists checked images from a patient's record of previous EGDs before performing a new EGD. In principle, the endoscopists including trainees performed the EGD alone. We focused on the latest EGD. The two last EGDs were analyzed for patients with non-detected GC. The EGD immediately before and at the time of cancer detection was analyzed for patients with detected GC. We defined the "same endoscopist" group as those who had the same endoscopist for the last two EGDs and the "different endoscopist" group as those who had different endoscopists for the last EGDs, regardless of whether GC was detected or not.

Endoscope type

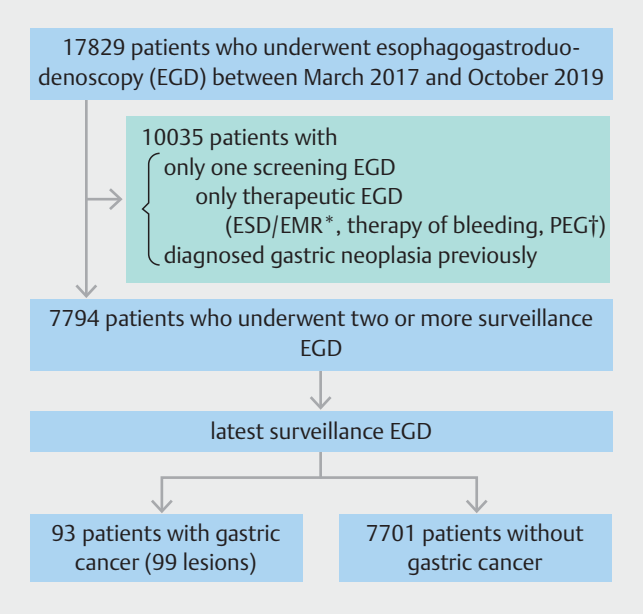
We used high-definition and non-high-definition endoscopes for surveillance EGD. The high-definition endoscopes were GIF-H290, GIF-H260Z, GIF-H290Z (Olympus, Tokyo, Japan), and EG-L600ZW7 (FUJIFILM, Tokyo, Japan). The non-high-definition endoscopes were GIF-XP260N, GIF-XP290N GIF-PQ260 (Olympus), and EG-L580NW7 (FUJIFILM). Evis Lucera Elite (Olympus) and Lasareo 7000 (FUJIFILM) were used as processors. Patients were sedated (using midazolam or propofol) during EGD based on patient request or the endoscopist's decision for the patient's safety and pain relief. Safety during sedation was ensured over time by biomonitoring. Endoscopists with <10 years or ≥10 years of endoscopic experience were defined as non-experts (n = 12) or experts (n = 17), respectively [12]. All expert endoscopists were certified by the Japan Gastroenterological Endoscopy Society, and non-expert endoscopists were not certified. The observation procedure was left to the endoscopist.

Pathological evaluation

Post-treatment specimens were evaluated according to the 14th Japanese Classification of Gastric Carcinoma (3rd English edition) [20], and curability with endoscopic treatment was evaluated according to the Gastric Cancer Treatment Guidelines version 5, published in January 2018 [22]. Tubular adenocarcinoma and papillary adenocarcinoma were considered differentiated cancers, while poorly differentiated adenocarcinoma and signet-ring cell carcinoma were considered undifferentiated cancers. Curability was categorized as curative [eCuraA (curative section) and eCuraB (expanded indication) with a low metastasis risk] or non-curative [eCuraC (non-curative resection) and surgical cases] [22].

Statistical analysis

All statistical analyses were performed using JMP 13 (SAS Institute, Cary, North Carolina, United States). The student's *t*-test was used to analyze continuous variables and the χ2 test or Fisher's exact test was used to analyze categorical variables. The factors affecting GC detection by surveillance EGD were assessed using univariate and multivariable analyses. In multivariable logistic regression analysis, variables considered to be associated with the detection of GC (age, sex, co-existence of atrophic gastritis, previous GC history, sedation usage, scope type, same or different endoscopist on EGD, and endoscopic experience) were included. Odds ratios (ORs) and 95% confidence intervals (CIs) were determined for significant variables on both analyses. Propensity-score matching (PSM) was performed with logistic regression analysis for age, sex, surveillance objective (atrophic gastritis and GC history), sedation, scope, and the experience of the same and different endoscopists. The c-statistic (area under the curve) for evaluating the discrimination was calculated. PSM was performed using nearest-neighbor matching algorithm without replacement and 1:1 matching. A caliper width of 0.05 of the standard deviation of the logit of the PSM was used. $P<0.05$ was considered statistically significant.



▶ **Fig. 1** Flowchart of patient enrollment. ESD/EMR, endoscopic submucosal dissection/endoscopic mucosal resection; PEG, percutaneous endoscopic gastrostomy.

Results

Characteristics of patients on surveillance EGD

Of the 17,829 patients who received EGD during this study period, 7794 were included in this study (▶ **Fig. 1**). ▶ **Table 1** lists patient characteristics (total patients, patients examined by the same endoscopist and by different endoscopist). Of the included patients, 4605 (59.1%) were male, and 4839 (62.1%) were aged ≥65 years. For surveillance purposes, 5040 patients (64.7%) had atrophic gastritis and 547 (7.0%) had a GC history. During the latest EGD, 5911 patients (75.8%) were sedated, high-definition scopes were used in 5421 patients (69.6%), and experts screened 3578 patients (45.9%). In 6555 patients (79.1%), an endoscopist different from the previous one performed the EGD. The mean surveillance interval was 12.1 months.

GC clinicopathologic characteristics detected by surveillance EGD

GC was detected in 93 patients (1.2%) with 99 lesions. The GC characteristics are summarized in ▶ **Table 2**. The mean age of those with detected GC was 72.9 ± 7.9 years, and the mean surveillance interval was 11.2 ± 2.3 months. Of the 93 detected patients, 74 (79.6%) were male, 84 (90.4%) were *Helicobacter pylori*-infected (current and previously infected combined), and 61 (65.6%) had severe atrophic mucosa of the stomach. Of the 99 lesions, 64 (64.6%) were the depressed macroscopic type (0-IIc), 94 (94.9%) were differentiated, and the median tumor size was 10 mm (range, 3–45 mm). The tumor locations were relatively evenly distributed. Eighty-six (86.9%) lesions were intramucosal carcinomas, and eighty-seven (87.9%) were curatively resected by endoscopy, although one patient had an advanced cancer which invaded the subserosa.

Comparison clinicopathologic characteristics of detected GC between the same endoscopist and different endoscopist

The characteristics of GCs detected from EGDs performed by the same endoscopist compared to those performed by a different endoscopist are shown in ▶ **Table 2**. Of the total 99 lesions, the same endoscopists detected nine GCs (9.1%) and different endoscopists detected 90 GCs (90.9%). Characteristics of GC detected were compared between the two groups. There were no statistically significant differences in age, sex, *H. pylori* status, atrophic gastritis, tumor location, tumor circumference, histologic type, tumor size, depth, curability of treatment, and surveillance interval. Only the macroscopic type of tumor showed significant differences ($P<0.05$).

Factors associated with GC detection in surveillance EGD

The logistic analysis of factors associated with GC detection in surveillance EGD is shown in ▶ **Table 3**. In univariate logistic analysis, age ≥65 years (OR, 4.61; 95% CI, 2.45–8.67), male sex (OR, 2.72; 95% CI, 1.64–4.52), atrophic gastritis (OR, 4.12; 95% CI, 2.19–7.75), GC history (OR, 2.18; 95% CI, 1.21–3.94), and high-definition scope (OR, 5.45; 95% CI, 2.52–11.8) were significant factors of GC detection by surveillance EGD. Conversely, there were no significant differences in sedation usage, fixed/different endoscopist, or endoscopic experience. In multivariable logistic analysis, it was suggested that a different endoscopist (OR, 2.47; 95% CI, 1.21–4.93), age ≥65 years (OR, 2.84; 95% CI, 1.47–5.47), male sex (OR, 2.83; 95% CI,

▶ **Table 1** Characteristics of patients on surveillance esophagogastroduodenoscopy.

Characteristics	Total (n = 7794)	Same endoscopist (n = 1239)	Different endoscopist (n = 6555)
Mean age, years	66.5 ± 12.0	71.0 ± 10.7	67.0 ± 12.0
Age, n (%)			
▪ < 65 years	2955 (37.9)	292 (23.6)	2663 (40.6)
▪ ≥ 65 years	4839 (62.1)	947 (76.4)	3892 (59.4)
Sex, n (%)			
▪ Male	4605 (59.1)	712 (57.5)	3893 (59.4)
▪ Female	3189 (40.9)	527 (42.5)	2662 (40.6)
Objective for surveillance, n (%)			
▪ Reflux esophagitis	2196 (28.2)	304 (24.5)	1892 (28.9)
▪ Esophageal cancer	179 (2.3)	61 (4.9)	118 (1.8)
▪ Atrophic gastritis	5040 (64.7)	792 (63.9)	4248 (64.8)
▪ Gastric ulcer	1563 (20.1)	342 (27.6)	1221 (18.6)
▪ Gastric cancer	547 (7.0)	137 (11.1)	410 (6.3)
▪ Duodenal ulcer	492 (6.3)	87 (7.0)	405 (6.2)
▪ Duodenal cancer	32 (0.4)	11 (0.9)	21 (0.3)
▪ Submucosal tumor	686 (8.8)	107 (8.6)	579 (8.8)
Sedation, n (%)			
▪ Use	5897 (75.7)	951 (76.8)	4946 (75.5)
▪ Non-use	1897 (24.3)	288 (23.2)	1609 (24.5)
Scope, n (%)			
▪ High-definition	5339 (68.5)	1139 (91.9)	4200 (64.1)
▪ Non-high-definition	2455 (31.5)	100 (8.1)	2355 (35.9)
Endoscopist, n (%)			
▪ Same endoscopist	1239 (15.9)		
▪ Different endoscopist	6555 (79.1)	–	–
Endoscopic experience, n (%)			
▪ Expert	4711 (60.4)	828 (66.8)	3883 (59.2)
▪ Non-expert	3083 (39.6)	411 (33.2)	2672 (40.8)
Patients detected GC, n (%)	93 (1.2)	9 (0.7)	84 (1.2)
Mean surveillance interval, months	12.1 ± 3.9	12.0 ± 4.5	12.0 ± 3.7

GC, gastric cancer.

1.69–4.74), atrophic gastritis (OR, 2.79; 95% CI, 1.47–5.30), and high-definition scope (OR, 3.82; 95% CI, 1.69–8.60) were significant factors associated with GC detection by surveillance EGD. Sedation usage and endoscopic experience were not significantly different in the multivariable analysis.

Analysis after PSM

The comparison of patient characteristics between the different endoscopist and same endoscopist groups after PSM analysis is shown in ▶ **Table 4**. We compared 1235 patients whose surveillance EGDs were performed by a different endoscopist with control patients whose EGDs were performed by a same endoscopist. They were adjusted using PSM based on covariates of age, sex, atrophic gastritis, GC history, sedation usage, scope type, and endoscopic experience (c statics = 0.765).

▶ Table 2 Clinicopathologic characteristics of gastric cancer detected by surveillance esophagogastroduodenoscopy.

Characteristics	Total 99 lesions (n = 93)	Same endoscopist 9 lesions (n = 9)	Different endoscopist 90 lesions (n = 84)	*P* value
Mean age, years	72.9 ± 7.9	75 ± 8.6	73 ± 7.9	0.50
Males, n (%)	74 (79.6)	7 (77.8)	67 (79.8)	1
Helicobacter pylori status[1], n (%)				
▪ Current	14 (15.1)	2 (22.2)	12 (14.3)	0.59
▪ Post eradication	70 (75.3)	6 (66.7)	64 (76.2)	
▪ Unknown	9 (9.7)	1 (11.1)	8 (9.5)	
Atrophy gastritis, n (%)				
▪ Mild	3 (3.2)	0 (0.0)	3 (3.6)	0.64
▪ Moderate	28 (30.1)	4 (44.4)	24 (28.6)	
▪ Severe	61 (65.6)	5 (55.6)	56 (66.7)	
▪ Unknown	1 (1.1)	0 (0.0)	1 (1.2)	
Tumor location, n (%)				
▪ U	18 (18.2)	1 (11.1)	17 (18.9)	0.81
▪ M	35 (35.3)	4 (44.4)	31 (34.4)	
▪ L	46 (46.5)	4 (44.4)	42 (46.7)	
Tumor circumference, n (%)				
▪ Anterior wall	13 (13.2)	0 (0.0)	13 (14.4)	0.50
▪ Posterior wall	33 (33.3)	2 (22.2)	31 (34.4)	
▪ Lesser curvature	33 (33.3)	4 (44.4)	29 (32.2)	
▪ Greater curvature	20 (20.2)	3 (33.3)	17 (18.9)	
Macroscopic type, n (%)				
▪ 0-I	1 (1.0)	0 (0.0)	1 (1.1)	< 0.05
▪ 0-IIa	22 (22.2)	2 (22.2)	20 (22.2)	
▪ 0-IIb	7 (7.1)	2 (42.2)	5 (5.6)	
▪ 0-IIc	64 (64.6)	3 (33.3)	61 (67.8)	
▪ 0-IIa + IIc	5 (5.1)	2 (22.2)	3 (3.3)	
Histologic type, n (%)				
▪ Differentiated type	94 (94.9)	9 (100.0)	85 (94.4)	1
▪ Undifferentiated type	5 (5.1)	0 (0.0)	5 (5.6)	
Median tumor size, mm (range)	10 (3–45)	10 (7–28)	9 (3–45)	0.15
Depth, n (%)				
▪ M	86 (86.9)	8 (88.9)	78 (86.7)	1
▪ SM1	2 (2.0)	0 (0.0)	2 (2.2)	
▪ SM2	10 (10.1)	1 (11.1)	9 (10.0)	
▪ MP	0 (0.0)	0 (0.0)	0 (0.0)	
▪ SS	1 (1.0)	0 (0.0)	1 (1.1)	

▶ Table 2 (Continuation)

Characteristics	Total 99 lesions (n = 93)	Same endoscopist 9 lesions (n = 9)	Different endoscopist 90 lesions (n = 84)	*P* value
Curability of treatment, n (%)				
▪ Curative	87 (87.9)	8 (88.9)	79 (87.8)	1
▪ Non-curative	12 (12.1)	1 (11.1)	11 (12.2)	
Mean surveillance interval, months	11.2 ± 2.3	12.0 ± 3.9	12.0 ± 2.4	0.36

M, mucosa; SM, submucosa; MP, muscularis propria; SS, subserosa.
SM1 is an invasion depth of less than 500 μm into the submucosa and SM2 is 500 μm or more.
[1] Current: currently infected with *Helicobacter pylori*; Post: After *H. pylori* eradication

▶ **Table 3** Factors associated with gastric cancer detection in surveillance esophagogastroduodenoscopy.

	Univariate Analysis OR (95 % CI)	*P* value	Multivariable analysis OR (95 % CI)	*P* value
Age				
▪ < 65 years	Reference	< 0.05	Reference	< 0.05
▪ ≥ 65 years	4.61 (2.45–8.67)		2.84 (1.47–5.47)	
Sex				
▪ Male	2.72 (1.64–4.52)	< 0.05	2.83 (1.69–4.74)	< 0.05
▪ Female	Reference		Reference	
Coexistence of atrophic gastritis				
▪ Yes	4.12 (2.19–7.75)	< 0.05	2.79 (1.47–5.30)	< 0.05
▪ No	Reference		Reference	
Previous history of GC				
▪ Yes	2.18 (1.21–3.94)	< 0.05	1.31 (0.72–2.41)	0.37
▪ No	Reference		Reference	
Sedation				
▪ Use	1.16 (0.71–1.92)	0.63	1.18 (0.71–1.99)	0.52
▪ Non-use	Reference		Reference	
Scope				
▪ High-definition	5.45 (2.52–11.8)	< 0.05	3.82 (1.69–8.60)	< 0.05
▪ Non-high-definition	Reference		Reference	
Endoscopist				
▪ Same endoscopist	Reference	0.10	Reference	< 0.05
▪ Different endoscopist	1.77 (0.89–3.54)		2.47 (1.21–4.93)	
Endoscopic experience				
▪ Expert	1.06 (0.70–1.59)	0.83	1.22 (0.80–1.85)	0.35
▪ Non-expert	Reference		Reference	

OR, odds ratio; CI, confidence interval; GC, gastric cancer.

▶ **Table 4** Characteristics of patients after propensity-score matching analysis to compare the use of a same endoscopist and different endoscopists.

Characteristics	Same endoscopist (n = 1235)	Different endoscopist (n = 1235)	*P* value
Age, n (%)			
▪ < 65 years	292 (23.6)	291 (23.6)	0.96
▪ ≥ 65 years	943 (76.4)	944 (76.4)	
Sex, n (%)			
▪ Male	709 (57.4)	706 (57.2)	0.90
▪ Fmale	526 (42.6)	529 (42.8)	
Coexistence of atrophic gastritis, n (%)			
▪ Yes	792 (64.1)	795 (64.4)	0.90
▪ No	443 (35.9)	440 (35.6)	
Previous history of GC, n (%)			
▪ Yes	133 (10.8)	132 (10.7)	0.95
▪ No	1102 (89.2)	1103 (89.3)	
Detected GC, n (%)	9 (0.7)	20 (1.6)	< 0.05
Sedation, n (%)			
▪ Use	973 (78.8)	975 (79.0)	0.92
▪ Non-use	262 (21.2)	260 (21.0)	
Scope, n (%)			
▪ High-definition	1147 (92.9)	1148 (93.0)	0.94
▪ Non-high-definition	88 (7.1)	87 (7.0)	
Endoscopic experience, n (%)			
▪ Expert	812 (65.7)	811 (65.7)	0.97
▪ Non-expert	423 (34.3)	424 (34.3)	

GC, gastric cancer.

There were 9 (0.7 %) GCs detected by same endoscopists and 20 (1.6 %) detected by different endoscopists ($P = 0.04$, $\chi 2$ test). All matched factors after PSM were well-balanced in this study. Age, atrophic gastritis, sedation usage, type of scope, and endoscopic experience were not significantly different in the univariate analysis after PSM.

Discussion

In this study, we identified the key factors for GC detection in view of surveillance EGD by analyzing results of more than 7,000 EGD screenings. The frequency of GC detection was higher when different endoscopists performed consecutive EGDs than when the same endoscopist performed them on a given patient. The present study suggests that assigning a different examiner each year might be an effective and simple strategy for improving GC detection through surveillance EGDs without additional cost.

GC detected by surveillance EGD

The protocol for surveillance EGD for GC recommends annual follow-up [7], and GC detected within the follow-up interval is assumed to have been missed. The GC detection rate in screening EGD is reported to be 0.91 % [14]. In a meta-analysis, it was reported that the median occurrence of metachronous cancer was 2.9 % in *H. pylori*-positive individuals and 1.2 % after eradication therapy [23]. The rate of metachronous cancer after endoscopic treatment was reported as 2.0 % per year [8]. We had a GC detection rate of 1.2 %, which was similar to that of a previous study supporting surveillance EGD.

GC detection is often difficult due to the inflammation caused by *H. pylori* in the background mucosa and epithelium with low-grade atypia on the surface of GC after *H. pylori* eradication [24]. Although the average size of missed cancers has not been mentioned in many studies [9–11, 25, 26], Jin et al. reported that 87.4 % of GCs < 2 cm were detected by EGD within 2 years [27], and Abe et al. reported that a missed cancer is usually small (< 20 mm) and differentiated intramucosal cancer [28].

Similarly, heterotopic multiple gastric carcinomas had a reported median size of 10 mm (range, 1–50 mm) on an annual surveillance EGD [8]. In a Japanese cohort study, 95 % of GCs detected by annual surveillance EGD were endoscopically resectable [8]. In our study, surveillance EGD at a mean interval of 12 months detected a median size of 10 mm (range, 3–45 mm) as small, and 87.9 % of the patients were endoscopically curable. As previously reported, endoscopic accuracy was maintained. We recommend annual surveillance in high-risk populations for early detection and treatment. As indicated in ▶ **Table 2**, the only significantly different characteristic of GC detected in the same endoscopist and different endoscopist was the macroscopic type. This suggests that the factors contributing to GC detection may be related to factors in the endoscopic setting, rather than the GC itself. These 99 GCs had small tumors with a median size of 10 mm (range 3–45 mm). We retrospectively evaluated previous EGD images, but many of them could not be detected or were not in the EGD images. However, some of the large lesions and those that were deeply invasive GCs could be identified in the previous EGD images. This suggested that whether or not the presence of GCs was detected might be largely due to factors on the endoscopist's side, such as the observation procedure.

Improving the GC detection rate

The quality of EGD influences GC detection. There is limited evidence concerning EGD quality improvement methods. In a previous study, SSS [13], training [15], endoscopist experience [12, 16], longer examination time [14, 29], and the use of high-definition scopes [10] were reported as ways to increase the GC detection rate. Generally, the collective intelligence of multiple physicians can improve the diagnostic accuracy [17]. In colonoscopy, it was reported that adenoma detection rates are improved with multiple observations [18]. To the best of our knowledge, there have been few studies on whether same or different endoscopists contribute to the GC detection rate in surveillance EGD. In the present study, we found that changing the endoscopist remained a contributing factor toward GC detection. After PSM, the use of a different endoscopist had a significantly higher GC detection rate (1.6 %) than with a same endoscopist (0.7 %), indicating that changing the endoscopist can be a simple and effective way of increasing the GC detection rate in EGD screening. Occasionally, other lesions are detected during a repeat endoscopy of a GC lesion referred from another hospital. This may be due to the differences in endoscopic observation procedures, time, and the fact that some people are better or worse at observing certain parts, in addition to differences in modality. In this study, there were no significant differences in the characteristics of GCs detected among the two groups. We suspect that the different endoscopist group had a better detection rate for GC than the same endoscopist group because having a different endoscopist may correct for individual differences in endoscopic observation techniques, including procedures, time, and skill. However, depending on the number of endoscopists, the size of the institution, healthcare resources, and patient backgrounds, there may be no choice but to use the same endoscopist. Therefore, changing the endoscopist may not be applicable to all examinations and facilities. In situations where EGD with a change in endoscopist is possible, it may be recommended to improve the detection rate without additional costs.

In surveillance EGD, significant factors related to GC detection in multivariable analysis were age (≥ 65 years), male sex, background mucosal atrophy, use of a high-definition scope, and the use of different endoscopists. Older age (≥ 65 years), male sex, and background atrophic mucosa are known risk factors for GC [8, 30]. The use of high-definition scopes has been reported to contribute to GC detection [26], and this study supports those results. When a background-matched PSM analysis was performed to eliminate the influence of these factors, the different endoscopist group had a significantly higher GC detection rate. We consider this to be a simple and useful method that contributes to improved GC detection.

Park et al. reported that highly experienced endoscopists performed the exam for GC detection [26], but in a multicenter cohort study, the endoscopists' experience did not make a difference in GC detection [9]. In this study, the examiner's expertise was not a factor that contributed GC detection. The lack of a significant difference in GC detection rates between the expert and non-expert examiners even after PSM may have been due to the inappropriate separation of the two groups using 10 years of experience as a cut-off. However, it is possible for non-experts to detect GCs, and we considered the possibility that individual differences in observation procedures, time, and individual peculiarities may have caused the difference, more than the cut-off problem. In a national Delphi survey of endoscopy education, 10 competencies have been identified that are essential for endoscopy education [31]. One of the essential competencies is that the experts should be able to control the procedure when trainees are unable to progress. This competency is related to the fact that collective intelligence can improve the diagnostic accuracy [17]. Regardless of endoscopic experience, our results suggest that trainees may be able to perform an examination without reducing the detection rate by changing the endoscopist for each exam with appropriate education. This simple method may also contribute to endoscopy education.

Limitations

This study has several limitations. First, this was a single-center, retrospective study. Second, although there was no significant difference, there were more EGDs performed by non-experts because of endoscopic training. Third, the ability to detect GC was not quantifiable. Based on a previous study [12], we divided all examiners into two categories: non-experts with < 10 years of experience and experts with ≥ 10 years of experience. There is room for reconsideration of how to divide the two categories, as both trainees and experts with more than 20 years of experience were included. A particular problem was that non-experts included a small number of trainees who needed supervisors for endoscopy, as well as endoscopists with skills close to those of experts. Fourth, the background of patients for surveillance EGD included other cases with factors other than atrophic gastritis, intestinal epithelialization [32], and history of GC [7],

which are commonly referred to as high-risk factors of GC. In addition, smoking [33], genetic background and family history [34], and some *H. pylori* statuses were not investigated. If the focus is only on the occurrence of GC, the number of cases should be further narrowed (e. g., excluding cases with mild atrophy). Fifth, although atrophic gastritis was determined endoscopically using the Kimura-Takemoto classification [19], pathological evaluation should have been performed using the Sydney classification [35] to accurately assess the presence or absence of severe atrophy or intestinal epithelialization, which is a high-risk factor for GC. Sixth, all endoscopic images could not be reevaluated for mucosal atrophy by a single endoscopist; therefore, the study could not be tailored to the GC risk of mucosal atrophy grade.

Conclusions

In conclusion, on performing surveillance EGD every 12 months, GC was detected in 1.2 % of patients. Regardless of endoscopic experience, changing the endoscopist for each exam may contribute to improving the GC detection rate by correcting individual differences.

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Competing interests

The authors declare that they have no conflict of interest.

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