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Clinical Outcomes Based on the Timing of Appearance of Visible Lesions in Barrett's Esophagus During Endoscopic Eradication Therapy

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Abstract

Background and Goals: Visible lesions (VLs) in Barrett's esophagus (BE) are seen in 70% to 90% of patients presenting for endoscopic eradication therapy (EET). It is not known if there are any differences in outcomes of patients with flat dysplasia versus patients with VL. Our aim was to assess outcomes of EET in BE patients with VL and BE patients with flat dysplasia.

Study: This is a single center study with data drawn from a prospective registry of patients referred for EET of BE between 2011 and 2015. Demographic data, endoscopic findings, histologic findings, and response to EET were analyzed.

Results: There were 264 patients of which 34 had flat dysplasia, 180 had VL before initiating EET (prevalent lesions) and 50 who developed VL during EET (incident lesions). Compared with patients with flat dysplasia, patients with VL had longer segments of BE (5 vs. 4cm, $P=0.002$) and greater prevalence of high-grade dysplasia (HGD) or esophageal adenocarcinoma (EAC) (63.6% vs. 29.4%, $P<0.001$). Incident lesions are less likely to harbor HGD/EAC compared with prevalent lesions (28.1% vs. 61.8%, $P<0.001$). There were no significant differences in eradication of metaplasia/dysplasia between the groups. No progression or recurrences were observed in flat dysplasia group. In VL group, 14 patients progressed (prevalent VL=11, incident VL=3) and 15 had recurrences (prevalent VL=11, incident VL=4).

Conclusions: About 19% of BE patients developed VL during EET. There is higher prevalence of HGD/EAC in prevalent VL compared with incident VL. However, the outcomes did not differ.

Keywords

Barrett's esophagus; visible lesions; outcomes; endoscopic therapy; esophageal cancer

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The authors declare that they have nothing to disclose.

The first step in the neoplastic transformation of Barrett's esophagus (BE) is a change in the cytological features and architecture of metaplastic glands, which manifests histologically as dysplasia. Dysplasia may sometimes present as subtle abnormalities visible endoscopically within the BE segment. These visible lesions (VLs) include nodules, ulcers, plaques, strictures, or polypoid lesions. Presence of these lesions are known to be associated with prevalent high-grade dysplasia (HGD) or esophageal adenocarcinoma (EAC).^{1,2} Hence, all the current guidelines on management of BE recommend targeted biopsy of any VLs in addition to systematic 4 quadrant biopsies of the BE segment every 1 to 2cm intervals. A forceps biopsy is not adequate to provide complete histologic assessment and when feasible, endoscopic mucosal resection (EMR) should be performed. EMR leads to a change in diagnosis in up to 50% of patients.³ EMR not only serves as a therapeutic modality, but also provides tissue for accurate histopathologic assessment which guides further therapy. As currently available ablation techniques may not adequately penetrate the entire depth of dysplastic tissue, EMR of any VLs for accurate staging and complete removal followed by ablation of remaining BE segment have become the standard of therapy for BE-associated neoplasia.

Accurate detection of these subtle changes can be challenging. Between 50% and 90% of BE patients referred for endoscopic eradication therapy (EET) are found to have VLs by expert endoscopists, not reported in prior endoscopy.⁴ Identification of those lesions and adequate resection are essential for successful eradication of BE. However, the impact of the detection of VLs on clinical outcomes is not clearly known and currently available evidence is somewhat contradictory. Studies with high rate of detection of VLs and subsequent EMR report eradication rates >90%, whereas those with EMR in <50% of patients report lower eradication rates (~60%).^{5,6} In contrast, large US-based studies report lack of effect of preablative EMR for VLs on the complete eradication of intestinal metaplasia (CE-IM) and dysplasia (CE-D).^{7,8} Another issue is development of metachronous lesions which are reported to occur in 4% to 28% of patients during EET of BE.^{9,10} Except for one study in which 4% of these lesions were submucosal cancers, the impact of these metachronous lesions on clinical outcomes is not known.⁹

Therefore, the goals of our study were: (1) to assess progression during EET and recurrence after successful EET in patients presenting with VLs versus flat dysplasia, (2) to study any difference in outcomes in patients presenting with VLs before EET (prevalent lesions) versus VLs which develop during EET (incident lesions) and (3) to assess eradication rates of metaplasia and dysplasia in patients with VLs versus those with flat dysplasia.

METHODS

Study Population

Patients with BE referred for EET at our institution between January 1, 2011 to December 31, 2015 were included in the study. Demographic data such as age, gender, race, and endoscopic data including length of BE segment, hiatal hernia size, endoscopic appearance of VLs and endoscopic therapy and histologic data were included. The data were collected as a part of BE registry and approved by our Institutional Review Board.

Treatment Protocol

Before initiating EET, a detailed endoscopic examination was performed with high definition white light endoscopy and narrow band imaging, with particular attention to presence and endoscopic appearance of VLs. Endosonographic examination was also performed based on endoscopists' discretion. The BE segment was described using Prague classification based on the location of endoscopic landmarks, such as the diaphragm, esophagogastric junction, and circumferential (C) and maximum (M) extent of the BE.¹¹ VLs were characterized as nodules, ulcers, masses, plaques, polypoid lesions, and mucosal abnormalities.¹² Four quadrant biopsies were obtained every 1cm interval if not performed during prior endoscopy. Any VLs were resected when feasible by EMR. If there were no VLs, radiofrequency ablation (RFA) was performed with RFA-360 catheter if BE segment was ≥ 3 cm or focal RFA 90 or 60 if BE < 3 cm. Patients were prescribed proton pump inhibitors twice daily, Sucralfate 1g 4 times a day for 10 days and cocktail consisting of diphenhydramine, aluminium and magnesium hydroxide, and lidocaine. Patients were instructed to return every 2 to 3 months until all endoscopically visible BE was eradicated. VLs which developed during EET (incident lesions) or BE persistent after multiple sessions of RFA were treated by EMR. Patients with progression were discussed at a multidisciplinary meeting for decision on further management, depending on operative risk and life expectancy.

Patients with CE-IM or CE-D patients were advised to return for surveillance endoscopies at intervals based upon the grade of dysplasia which prompted EET. If the patient had HGD or EAC before ablation, they had surveillance endoscopies every 3 months for the first year, every 6 months in the second year, and then every year thereafter. During surveillance endoscopies, 4 quadrant biopsies were performed from gastric cardia and original BE segment at 1cm intervals.

Histology

In cases of dysplasia or EAC, diagnosis was confirmed by a gastrointestinal pathologist or reviewed at a consensus conference. The biopsy results were reported with no dysplasia, low-grade dysplasia (LGD), indefinite for dysplasia, HGD, or EAC as per Vienna classification.¹³ The worst histologic grade identified was taken as the overall histologic grade for that endoscopy. The histologic diagnosis for VL and flat mucosa were reported separately. Histology from EMR specimen when available is used for statistical analysis.

Outcomes

Patients found to have submucosal disease or worse on initial EMR were not considered as candidates for EET and therefore were not included for calculation of clinical outcomes. The following outcomes were assessed:

1. CE-IM is defined as absence of any visible BE on endoscopy and intestinal metaplasia on first surveillance biopsies after EET.
2. CE-D is defined as persistent metaplasia with absence of any dysplasia on histology. It may or may not be associated with endoscopic BE.

3. Progression is defined as higher grade of dysplasia compared with index biopsy anytime during EET.
4. Recurrence is reappearance of intestinal metaplasia or dysplasia after prior CE-IM or dysplasia after CE-D.

Statistical Analysis

Patients were divided into 3 groups: (1) flat dysplasia: patients did not have any VLs at presentation or during the study period; (2) patients with prevalent VLs, and (3) patients with incident VLs. In patients with >1 lesion on the same endoscopy, the lesion with the worst pathology was considered as first lesion and the other was counted as second lesion. Data are presented as mean±SD, median (25th, 75th percentiles) or frequency (percent). Univariable analysis was performed to assess differences between the subject groups (no VLs, lesions before treatment and lesions after treatment). Analysis of variance or the nonparametric Kruskal-Wallis tests were used to compare continuous variables and Pearson's χ^2 tests or the Fisher Exact tests were used for categorical factors. Follow-up time was defined as months from the index endoscopy to the first endoscopy showing progression or last endoscopy if no progression was seen. A time to event analysis was performed to assess progression rates and recurrence rates. Kaplan-Meier plots were constructed and log-rank tests were used to compare the groups. All analyses were performed using SAS (version 9.4; The SAS Institute, Cary, NC) and a $P < 0.05$ was considered statistically significant.

RESULTS

The cohort consisted of 264 patients with a median age of 64 years [interquartile range (IQR), 57 to 72] and predominantly white (255; 97%). Male/female distribution was 232 to 32. The median size of the hiatal hernia was 3 cm (IQR, 2 to 4) and length of BE segment was 4 cm [IQR, 2 to 8]. The median follow-up was 27.6 months [IQR, 12 to 58.8]. The flowchart is presented in Figure 1.

VLs Versus Flat Dysplasia

There were 230 patients in the VLs group, of which 180 patients presented with VLs before EET (prevalent lesions) and 50 patients developed VLs after initiation of EET (incident lesions). The median time for the development of incident lesions was 7.6 months with 16% developing within 3 months, 24% between 3 and 6 months, 22% between 6 and 9 months, 12% between 9 and 12 months, and 26% after 12 months. Thirty-four patients never had any VLs during the study period and were included in the flat dysplasia group (Table 1). Patients with VLs had longer BE segments. In the flat dysplasia group, 4 patients with nondysplastic BE were referred for EET due to family history of EAC; most patients had LGD and none had EAC.

In the VL group, 5 patients did not have any intestinal metaplasia on random biopsies, but had HGD on histology of VLs. Twelve patients had nondysplastic BE in both VLs and on biopsy of flat mucosa and 13 patients had nondysplastic BE on random biopsies but dysplasia on VL histology (LGD in 2, HGD in 8, and EAC in 3). There was 1 patient with

squamous cell cancer within BE segment. Multifocal HGD or EAC was observed only in VL group. Among all VLs, the prevalence of HGD/EAC was 66.7% in nodular/polypoid lesions, 38.7% in ulcerated lesions and 25% in strictures.

Prevalent VLs Versus Incident VLs

There was higher prevalence of HGD/EAC in the VLs in the prevalent group compared with the incident group (Table 2). However, there was a higher prevalence of HGD/EAC in the flat mucosa of incident group compared with the prevalent group. More than one VL was noted in 20 patients (Table 2). EMR was performed in 20 of incident lesions. In the remaining incident lesions, specimen was obtained by biopsy.

Clinical Outcomes

In patients presenting with flat dysplasia (n=34), CE-IM was achieved in 19 and CE-D in 27 patients (Table 3). None of the patients progressed or developed recurrences during follow-up (Figs. 2, 3). Four patients did not return for treatment and 3 patients were undergoing treatment at the end of study period.

In patients presenting with prevalent VLs (n=180), 22 patients had advanced disease on EMR and were not deemed to be candidates for EET. They were referred for further therapy such as esophagectomy or chemoradiation therapy depending on the operative risk. CE-IM was achieved in 102 patients and CE-D in 131 patients (Table 4). Eleven patients progressed (Fig. 2). Ten patients did not return for further treatment sessions and 2 patients were undergoing treatment at end of study period. There was recurrent disease in 11 patients, of which 8 had nondysplastic BE, 1 developed LGD, 1 HGD, and 1 advanced cancer (Fig. 3). Four patients died during ongoing EET due to unrelated causes.

In patients who developed incident lesions during EET (n=50), 36 patients had CE-IM and 46 patients had CE-D. Random biopsies showed higher prevalence of HGD/EAC in the incident lesions group than the prevalent lesions group (Table 2). This is an interesting finding and likely due to missed lesions during index endoscopy which became more noticeable during treatment course. Interestingly, they did not require more treatment sessions than patients with prevalent lesions (Table 4). Three patients progressed to worse disease: 1 underwent esophagectomy, 1 went for radiation, and third died of amyotrophic lateral sclerosis (Fig. 2). One patient did not return for EET at the end of follow-up. Four patients developed recurrences after CE-IM and all were nondysplastic BE (Fig. 3). Outcomes based on VL type are presented in Table 5.

DISCUSSION

Our large single center study examined the effect of VLs on the outcomes in patients referred for EET of BE. Approximately 20% of the patients developed VLs during EET. We observed that progression during EET and recurrences after successful CE-IM were observed only in patients with VLs. There were no differences in the CE-IM and CE-D between patients presenting with flat dysplasia versus those with VLs. In addition, the timing of appearance of VLs does not appear to affect eradication rates.

We observed a lack of progression or recurrences in patients with flat dysplasia. These findings are somewhat contradictory to prior studies from United States reporting on the effect of preablative EMR (of prevalent lesions) on recurrence and progression. There was no mention of VLs in these studies, but reporting of preablative EMR is used as an inference for presence of VLs.^{7,8} In US RFA registry, neoplastic progression was observed in 1.5% of patients with VLs compared with 3.6% of patients who underwent RFA only (therefore, presumably had flat dysplasia only).⁷ Among the patients who achieved CE-IM, 57 of 277 patients (21%) who were treated with EMR before RFA for nodular BE, and 108 of 554 patients (19%) who were treated with RFA alone developed recurrence of BE.⁷ In a US multicenter study of 192 patients who were undergoing surveillance, 37 patients developed recurrence.⁸ EMR before RFA was not predictive of recurrence [EMR before RFA vs. no EMR; hazard ratio (HR), 1.18 (0.6, 2.34); $P=0.62$]. The plausible reasons for the differences between our study and other studies may be due to the predominance of LGD in our flat dysplasia group compared with a 70% to 100% prevalence of HGD or EAC in the other 2 studies and also due to the fact that the presence of VLs was not the primary focus of those studies.

The appearance of incident lesions during EET have been described in prior studies but their effect on the EET outcome was not systematically evaluated except for in 1 study. In a UK study of 50 patients referred for RFA of which 76% had prior EMR for a VL, 20 incident lesions were detected in 14 patients of which HGD was seen in 11, intramucosal cancer in 5, and submucosal cancer in 2 lesions.¹⁰ In Euro II trial of 132 patients, incident lesions during EET were reported in 6 patients, of which 1 was found to have submucosal cancer.⁵ In UK RFA registry of 335 patients undergoing RFA, incident lesions were reported in 10% of the patients.⁶ The varied rates may be due to differences in study design (clinical trial vs. observational) and diverse study populations. In our study, we observed that 18.9% patients developed incident lesions during EET, of which 9 had HGD/EAC but did not require more treatment sessions compared with flat dysplasia or prevalent lesions. Moreover, the risk of progression and recurrences were similar to the group presenting with prevalent lesions.

In this study, we observed that all patients with EAC had VLs and very few patients with HGD had flat BE. This is similar to the Amsterdam group findings which reported that a diagnosis of HGD or EAC is most likely to be associated with VLs.⁴ This is in contrast to US RFA registry where upto 76% of HGD and 26.3% of intramucosal cancers did not have a VL on presentation.⁷ Second, we observed that the dysplasia grade depends on endoscopic appearance of lesion: nodular lesions have higher prevalence of HGD/EAC than ulcers.

The identification of VLs and their removal is crucial for optimal outcome of EET. In cohorts where there is high recognition of VLs and therefore a frequent use of EMR, CE-D, and CE-IM are high. This is reflected in Euro II trial where up to 90% had a VL detected and EMR performed and CE-IM was seen in 87% and CE-neoplasia in 92%.⁵ After a median of 27 months following the first negative posttreatment endoscopic control, neoplasia and intestinal metaplasia recurred in 4% and 8%, respectively. In contrast, the reported rates of CE-IM and CE-D are low where EMR is not as frequent. For example, in UK RFA registry where preablation EMR was performed in 49%, CE-IM was seen in 62% and CE-D in 81%.⁶ In that study, 33 patients (10%) required rescue EMR for metachronous

lesions and 10 patients (3%) progressed to cancer at 1 year. However, in other studies, preablative EMR does not appear to have any effect on CE-IM or CE-D.^{7,8}

One of the point we would like to highlight here is that we included strictures and erosive esophagitis in the VLs group even though they are not VLs per se as they are markers for the presence of dysplasia and denote an increased risk of progression. In a study by Hillman et al,¹⁴ 353 patients undergoing surveillance were evaluated for macroscopic markers: severe esophagitis, ulcer, nodularity, and stricture. On median follow-up of 42 months, 13 patients developed HGD or EAC. Twelve of these 13 patients had 1 of these markers. Patients with 1 marker were 6.7 times more likely to develop HGD/EAC [HR=6.7; 95% confidence interval (CI)=1.3, 35] and those with 2 markers were 14 times more likely to develop HGD/EAC (HR=14.1; 95% CI=2.02, 102). Evidence also comes from another study where erosive esophagitis increased the risk of recurrence in BE patients undergoing EET on multivariable regression analysis controlling for hiatal hernia length and presence of dysplasia (HR=15.41; 95% CI, 1.65, 144.33).¹⁵ In addition, presence of a stricture may interfere with optimal cooptation against mucosa with ablation device and may affect the eradication rates.

The main strength of this study is that it is performed in a tertiary care center with specialized expertise in evaluation and treatment of BE-associated neoplasia. All pathology specimens were reviewed and confirmed by experienced gastrointestinal pathologists at our institution. There are several limitations to this study. First, Paris classification was not consistently used to describe all lesions. There are missing histologic findings for certain VLs such as erosive esophagitis and strictures. Another limitation is that the median follow-up period is only 28 months, which may not be adequate as recurrences have been reported for up to 5 years after ablation therapy.⁹ However, as per the AIM dysplasia trial, most recurrences were in the first year and rate of recurrence is not constant.¹⁶ The lack of differences in eradication rates may be due to a type II error, but similar findings have been observed in US RFA registry data.⁷ Finally, some subjects identified as having recurrence may have had incomplete treatment in the first place rather than a true recurrence.

In conclusion, VLs develop in approximately one-fifth of the patients during EET. The presence of VLs and timing of appearance of VLs during EET do not appear to impact eradication rates in patients with BE undergoing EET. Moreover, in patients presenting with flat dysplasia, recurrences or progression have not been observed. Larger studies and longer follow-up periods are needed to confirm these findings.

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FIGURE 1.

Flow chart of study patients. CE-D indicates complete eradication of dysplasia; CE-IM, complete eradication of intestinal metaplasia; EET, endoscopic eradication therapy.

FIGURE 2.
Progression rates in the study groups.

FIGURE 3.
Recurrence rates in the study groups.

TABLE 1.**Clinical Features and Histologic Findings in Study Population**

Factors	No Visible Lesions (N=34)	Visible Lesions (N=230)	P
Age (y)	63.5 (55.0, 73.0)	63.5 (55.0, 71.0)	0.58 ^a
Male gender	28 (82.4)	204 (88.7)	0.29 ^b
Race			
Caucasian	34 (100.0)	221 (96.5)	0.75 ^b
African American	0 (0)	6 (2.6)	
Hiatal hernia size (cm)	2.0 (1.00, 4.0)	3.0 (2.0, 4.0)	0.30 ^a
Length of BE (cm)	4.0 (1.00, 4.0)	5.0 (3.0, 8.0)	0.002 ^a
Prague C	2.0 (0.00, 3.0)	4.0 (2.0, 8.0)	0.005 ^a
Prague M	4.0 (1.00, 4.0)	6.0 (3.0, 9.0)	0.008 ^a
Index biopsy of flat mucosa			<0.001 ^b
No metaplasia	0 (0)	5 (3.5)	
Nondysplastic BE	4 (11.8)	25 (13.0)	
LGD	20 (58.8)	41 (20.8)	
HGD	10 (29.4)	99 (51.6)	
EAC	0 (0.0)	22 (11.5)	
Multifocal LGD	8	12	
Multifocal HGD	0	14	
Multifocal cancer	0	1	

Statistics presented as Mean±SD, Median (P25, P75), or N (column %). BE indicates Barrett's esophagus; EAC, esophageal adenocarcinoma; HGD, high-grade dysplasia; LGD, low-grade dysplasia.

P values

^aKruskal-Wallis test.

^bPearson χ^2 test.

TABLE 2.**Clinical Features and Histologic Findings in Visible Lesions Group**

Factors	Prevalent Lesions (N=180)	Incident Lesions (N=50)	P
Age (y)	64 (56, 71)	63 (58, 72)	0.98 ^a
Male gender	160 (88.9)	44 (88)	0.86 ^a
Race			
White	174 (97.2)	47 (94)	0.22 ^b
African American	4 (2.2)	2 (4)	
Hiatal hernia size (cm)	3.0 (2.0, 4.0)	3.0 (2.0, 4.0)	0.91 ^a
Length of BE (cm)	5.0 (2.0, 8.0)	6.0 (3.0, 8.0)	0.48 ^a
Prague C	4.0 (2.0, 7.0)	6.0 (2.0, 9.0)	0.33 ^a
Prague M	6.0 (3.0, 9.0)	8.0 (3.0, 9.0)	0.43 ^a
No. visible lesions			
1	164 (91.1)	46 (92)	0.84 ^a
2	15 (8.3)	4 (8.0)	
3	1 (0.56)	0 (0)	
Endoscopic appearance			
Mass	12	0	
Nodule	103	22	
Polypoid	9	0	
Erosive esophagitis	14	2	
Ulcerated	25	11	
Strictures	8	11	
Unspecified	9	4	
Visible lesion histology			<0.001
No metaplasia	18 (12.5)	16 (50)	
Nondysplastic BE	19 (13.2)	3 (9.4)	
LGD	17 (11.8)	4 (12.5)	
HGD	49 (34.0)	8 (25.0)	
Cancer	40 (27.8)	1 (3.1)	
Squamous cell cancer	1 (0.69)	0 (0.0)	
Endoscopic appearance of second lesion			0.75 ^b
Nodule	11 (68.8)	3 (75)	
Ulcer	3 (18.8)	1 (25)	
Others	2 (12.5)	0	
Histology of second lesion			0.92 ^b
No metaplasia	3 (21.4)	1 (25)	
Nondysplastic BE	6 (42.9)	1 (25)	
LGD	2 (14.3)	1 (25)	

Factors	Prevalent Lesions (N=180)	Incident Lesions (N=50)	P
HGD	3 (21.4)	1 (25)	
Random biopsies from flat mucosa			0.003 ^b
No metaplasia	5 (3.5)	0 (0.0)	
Nondysplastic BE	24 (16.8)	1 (2.0)	
LGD	27 (18.2)	14 (28.6)	
HGD	66 (46.2)	33 (67.3)	
Cancer	21 (14.7)	1 (2.0)	
Multifocal LGD	6 (23.1)	6 (42.9)	0.19 ^b
Multifocal HGD	10 (15.2)	4 (12.1)	0.68 ^b
Multifocal cancer	1 (4.8)	0 (0.0)	0.82 ^b

Statistics presented as Mean±SD, Median (P25, P75) or N (column %). BE indicates Barrett's esophagus; HGD; high-grade dysplasia; LGD, low-grade dysplasia.

P values

^aKruskal-Wallis test.

^bPearson χ^2 test.

TABLE 3.

Treatment Outcomes Depending on the Presence of Visible Lesions

Factors	Flat Dysplasia (N=27)	Visible Lesions (N=195)	P
Follow-up (mo)	29.7 (11.9, 54.4)	34.6 (15.6, 63.1)	0.27 ^b
No. EMR sessions	0	1 (0, 2)	<0.001 ^b
No. RFA sessions	2 (1, 3)	3 (2, 4)	0.089 ^b
No. EET sessions	2 (1, 3)	4 (3, 6)	<0.001 ^b
Eradication of metaplasia	20 (74.07)	138 (70.8)	0.84 ^a
Eradication of dysplasia	27 (100)	177 (90.3)	0.22 ^c
Progression	0	14 (7.2)	0.14 ^a
Recurrence	0	15 (8.5)	0.11 ^a

Patients who were deemed not eligible for endoscopic treatment, quit treatment, or were still undergoing treatment were excluded.

Statistics was presented as median (P25, P75) or N (column %).

EET indicates endoscopic eradication therapy; EMR, endoscopic mucosal resection; RFA, radiofrequency ablation.

P values

^a Analysis of variance.

^b Kruskal-Wallis test.

^c Pearson χ^2 test.

TABLE 4.

Outcomes Depending on the Timing of Appearance of Visible Lesions

Factors	Prevalent Lesions (N=146)	Incident Lesions (N=49)	
Follow-up (mo)	36.3 (14.0, 64.6)	32.3 (17.3, 59.1)	0.82 ^b
No. EMR	1.00 (1.00, 2.0)	1.00 (0.00, 1.00)	<0.001 ^b
No. RFA	3.0 (1.00, 4.0)	3.0 (3.0, 4.0)	0.011 ^b
Total No. EET	4.0 (3.0, 6.0)	4.0 (3.0, 5.0)	0.79 ^b
Eradication of metaplasia	102 (71.8)	36 (73.5)	0.82 ^a
Eradication of dysplasia	131 (92.2)	46 (93.9)	0.70 ^a
Progression	11 (7.5)	3 (6.1)	0.74 ^a
Recurrence	11 (8.5)	4 (8.7)	0.95 ^a

Patients who were deemed not eligible for endoscopic treatment, quit treatment, or were still undergoing treatment were excluded.

Statistics was presented as median (P25, P75) or N (column %).

EET, endoscopic eradication therapy; EMR indicates Endoscopic Mucosal Resection; RFA, Radiofrequency ablation.

P values

^a Pearson χ^2 test.

^b Kruskal-Wallis test.

TABLE 5.

Outcomes by Visible Lesion Type

Factors	Prevalent Lesions [n/N (%)]				Incident Lesions [n/N (%)]			
	Eradication of Metaplasia	Eradication of Dysplasia	Recurrence	Progression	Eradication of Metaplasia	Eradication of Dysplasia	Recurrence	Progression
Nodules	75/98 (76.5)	91/98 (92.8)	6/91 (6.5)	7/98 (7.14)	14/23 (60.86)	20/23 (86.95)	1/20 (5)	3/23 (13.04)
Polypoid	3/6 (50)	5/6 (83.3)	1/5 (20)	1/6 (16.6)	—	—	—	—
Ulcerated	14/18 (77.77)	17/18 (94.4)	1/17 (5.88)	1/18 (5.55)	7/10 (70)	10/10 (100)	2/10 (20)	—
Stricture	3/5 (60)	5/5 (100)	2/5 (40)	—	10/11 (90.9)	11/11 (100)	—	—
Esophagitis	3/9 (33.3)	8/9 (88.8)	—	1/9 (11.1)	2/2 (100)	—	—	—
Others	4/6 (66.6)	5/6 (83.3)	1/5 (20)	1/6 (16.6)	3/3 (100)	—	—	—