Barrett's esophagus is frequently overdiagnosed in clinical practice: results of the Barrett's Esophagus Endoscopic Revision (BEER) study CME

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Background: The published prevalence of Barrett's esophagus (BE) varies from 0.9% to 25%, in part because of differences in the endoscopic interpretation of the disease.

Objective: We studied the accuracy of diagnosis in 130 patients previously labeled as having BE. Our aim was to determine the interobserver consistency of endoscopic findings and assess the percentage of patients with confirmed BE versus those with a revised diagnosis.

Design/Setting/Patients: Patients previously diagnosed with BE of any length and due for surveillance endoscopy were eligible for study.

Interventions: After intensive consensus anatomic and endoscopic review, study patients underwent endoscopy and biopsy by 1 of 3 endoscopists. BE was defined as any length of columnar-lined esophagus with goblet cells.

Main Outcome Measurements: Patients were photographed/videotaped for review by the other 2 endoscopists, and BE was either confirmed or revised.

Results: Eighty-eight patients (67.7%) had confirmed BE, and 42 (32.3%) had their diagnosis revised to no BE (95% confidence interval, 24.4%-41.1%) because there was no visible columnar-lined esophagus proximal to the gastric folds or no goblet cells were found on biopsy. BE length, site of previous endoscopy, age, sex, and hiatal hernia size were predictors of revision. All 3 endoscopists agreed on all confirmed BE cases and 38 of 42 of those revised.

Limitations: Retrospective analysis, possible sampling error.

Conclusions: BE is overdiagnosed in clinical practice with important implications for patient care including increased costs, reduced insurability, and psychological stress. The true BE cancer risk may also be underestimated. This study suggests the need for a better definition of the gastroesophageal junction, stricter accountability for BE diagnosis, and improved endoscopic education. (Gastrointest Endosc 2014;79:565-73.)

Barrett's esophagus (BE) is conventionally defined as replacement of the squamous epithelium of the esophagus with columnar-type mucosa containing intestinal metaplasia.[1-3] As noted by the recent American Gastroenterology Association Institute (AGAI) Barrett's Esophagus Medical Position Statement, the presence of intestinal

Abbreviations: AGAI, American Gastroenterology Association Institute; BE, Barrett's esophagus; CI, confidence interval; GEJ, gastroesophageal junction; MN Gastro, Minnesota Gastroenterology; PPI, proton pump inhibitor.

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metaplasia is required for the diagnosis because, at present, intestinal metaplasia is the only type of columnar epithelium that clearly predisposes to malignancy.[1] Because of the considerable implications of finding BE, accurate endoscopic diagnosis is paramount; however, the AGAI also notes that current endoscopic definitions are problematic.[1] The region of the gastroesophageal junction (GEJ) is notoriously difficult to assess endoscopically as there are no universally accepted landmarks for discerning exactly where the esophagus ends and the stomach begins.[1,4]

Most gastroenterologists accept the top of the gastric folds as the main landmark of the GEJ; however, this anatomic border can be affected by the presence or absence of a sliding hiatal hernia, and air insufflation during endoscopy can flatten the folds, making it difficult to discern their proximal extent.[1,4] A high percentage of patients undergoing endoscopy, approximately 20%, can have intestinal metaplasia limited to the cardia portion of the stomach without columnar-lined epithelium proximal to the gastric folds.[5] These patients with so-called cardia intestinal metaplasia can be confused with having BE if the exact location of the top of the gastric folds is obscured. This is an important issue because biopsy specimens containing intestinal metaplasia arising from the region of the cardia are generally considered to be of little consequence, and biopsies proximal to this region yield a diagnosis of BE with its attendant consequences.[6]

Because of the definitional, anatomical, and histological issues described, perhaps not surprisingly, the published prevalence rates of BE vary considerably in the GI literature from, for example, 0.9% (Mayo Clinic autopsy study; minimal length defined), to 1.6% (prospective Swedish study; no length defined), to 6.8% (tandem colonoscopy/endoscopy study, Indiana University; no length defined), to 25% (tandem flexible sigmoidoscopy/endoscopy study, California VA, Los Angeles, CA; no length defined).[7-10] Although the studied populations differ, these discordant prevalence rates are problematic because they raise concerns about the reliability and interobserver consistency of a BE diagnosis. In addition, without accurate prevalence rates of BE, the true cancer risk cannot be determined. Those with BE have an esophageal adenocarcinoma progression rate that varies in the literature from 0.2% to 3.5% per year.[11-13] Recent studies report lower cancer progression rates; however, these studies are retrospective, assume a consistent and accurate BE endoscopic diagnosis at face value without verification, and use variable or no defined BE lengths.[12,13]

Given these factors, we believed that it was important to study the accuracy of a BE diagnosis in a population previously given a diagnosis of the disease. Surprisingly, there have been few studies that, via repeat endoscopy, have systematically assessed those previously labeled as having BE to verify the diagnosis and determine the percentage of diagnostic accuracy. We were interested in determining whether focused endoscopic training on foregut anatomy, in particular the anatomy of the GEJ, would allow interobserver consistency of endoscopic assessment, determine the percentage with confirmed BE, and determine what percentage of patients could have their diagnosis reversed altogether.

Take-home Message

- Barrett's esophagus is significantly overdiagnosed in clinical practice. This has broad implications for patient health and well-being, direct and indirect medical costs, and the ability to discern true cancer progression risk for Barrett's esophagus.

METHODS

Study design

The aim of the study was to determine the accuracy of the diagnosis of BE generated in routine endoscopic practice. This was done by performing a critical review of the initial diagnosis via follow-up endoscopy performed by a select small set of experienced, additionally trained endoscopists (ie, the study investigators). Findings at follow-up were compared with those in the initial examination. The main study outcomes were the number and percentage of BE patients with reversal of their diagnosis (no confirmed endoscopic columnar-lined esophagus proximal to the gastric folds or, if present, no confirmed intestinal metaplasia). The secondary outcomes of interest were to assess the interendoscopist revision rates and any predictors of revision. The study was conducted at the Minnesota Gastroenterology, PA (MN Gastro), outpatient ambulatory endoscopy center, located in Bloomington, MN, a suburb of Minneapolis. Minnesota Gastroenterology, PA, is a large, single-specialty gastroenterology private practice affiliated with the University of Minnesota.

Didactic training phase with nonstudy patients

Before enrolling study patients, from January to March 2010, the 3 investigators spent 8 hours together, first in didactic sessions, and then in joint endoscopic sessions, with 10 non-BE patients, reviewing GEJ anatomy and anatomic landmarks. Specifically, aspects of the tubular esophagus, the gastric cardia, the gastric folds, gastroesophageal junction both open and closed, the vascular rosette, hiatal hernia anatomy, Hill grade assessment of the cross-sectional area of the GEJ,[14] Los Angeles classification of esophagitis,[15] and the impact of air insufflation on the position of the z line and region of the GEJ were reviewed.

The 3 physicians then jointly performed endoscopy on 10 BE patients (these were not included in the study), to ensure agreement on landmarks and to refine any anatomic disagreements with regard to BE or the region of the GEJ. All patients were photographed and videotaped

to help with the training. The standard surveillance interval for patients with BE at Minnesota Gastroenterology, PA (Plymouth, Minn), is every 3 years, using Prague C&M diagnostic criteria[4] documented by a proprietary electronic medical record (NextGen, Horsham, Pa). The biopsy protocol is 4-quadrant biopsy samples taken every 2 cm, by using standard-size forceps (Conmed Precisor, Utica, NY).

Study population

The study cohort consisted of 130 patients who previously underwent endoscopy and received a diagnosis of BE, and were scheduled to return for routine surveillance endoscopy and biopsy to the Minnesota Gastroenterology, PA, outpatient ambulatory endoscopy center during the calendar years 2010 and 2011. To be included in the study, patients needed to have previously reported esophageal columnar-lined epithelium with intestinal metaplasia identified on a previous biopsy. Exclusion criteria included any previous esophageal or gastric surgery, any previous endoscopic treatment for GERD or BE, patients unable to undergo sedation for any reason, and those unable to understand the consent process. BE patients who previously underwent endoscopy by any of the investigators were also excluded. For data analysis purposes, the original endoscopic reports were reviewed, and patients were also classified as having previously received a diagnosis by a Minnesota Gastroenterology, PA endoscopist, or an endoscopist outside of the practice.

Follow-up expert endoscopy

Once included, patients underwent surveillance endoscopies as previously planned, but they were performed by a study investigator, with BE defined as any length of columnar-lined epithelium in the tubular esophagus proximal to the gastric folds and positive for intestinal metaplasia, with goblet cells on biopsy. Standard hematoxylin and eosin appearance was used for the recognition of goblet cells. Olympus 180 series endoscopes (Olympus, Allentown, Pa) were used, and narrow-band imaging was used in all cases. The biopsy protocol used for the study examinations was the same as that used for standard Minnesota Gastroenterology, PA, examinations as noted previously. The previous biopsy protocol for patients coming from providers who were not part of Minnesota Gastroenterology, PA, could not be assessed. All study specimens, including the presence of intestinal metaplasia, were reviewed and confirmed by at least 1 expert GI pathologist. The participating pathologists were well versed in the recognition of goblet cells; equivocal cases were adjudicated by consensus review by an additional similarly experienced pathologist.

At the study surveillance endoscopy, the previous diagnosis of BE was either confirmed (columnar mucosa was observed within the tubular esophagus and goblet cells were seen in the biopsy sample) or reversed. If confirmed, the length of BE was noted. Reversals fell into 3 categories: (1) patients with cardia intestinal metaplasia and no visible Barrett's esophagus; (2) patients with visible columnar-lined epithelium proximal to the gastric folds but no confirmatory intestinal metaplasia, ie, no goblet cells; and (3) patients with neither visible columnar-lined epithelium proximal to the gastric folds nor intestinal metaplasia.

Initial 30 patients

The study began in April 2010. Each of the 3 investigators independently performed endoscopy on 10 BE study patients (30 total) who were due for a routine, standard surveillance examination. Each endoscopist performed the examination in the usual manner, noting appropriate landmarks, assessing the GEJ, and assessing the presence or absence of BE. Each examination was videotaped, and pertinent areas of each examination, including any BE length and the area of the GEJ, were also photographed and stored for retrieval in the electronic medical record (NextGen) for later review. Standard endoscopic biopsy specimens were obtained (4-quadrant, every 2 cm) from the BE segment, if present, and biopsy specimens of the cardia region, at and just distal to the endoscopically determined squamocolumnar junction (z line), were obtained if BE was deemed to be absent. In every patient, 2 specimens each were also taken from the gastric body and gastric antrum to assess for possible *Helicobacter pylori*–induced intestinal metaplasia.

After each investigator's 10 procedures (30 total), the other 2 physicians independently reviewed the study materials (photographs, videotapes, histology) and either agreed or disagreed with the findings and final diagnosis. Any disagreements regarding anatomy or reversal of the BE diagnosis were adjudicated by joint discussion, review of the study materials, and majority (two thirds) vote.

100 Additional patients

Based on consistent assessments during the lead-in phase, the study continued with 100 additional previously diagnosed BE patients returning for routine surveillance examinations and studied in the same manner. Procedures were performed by 1 of the 3 investigators (photographed and videotaped), with the diagnosis of BE either confirmed or reversed by that investigator, and reviewed by the other 2 investigators. In this phase, all photographs were reviewed; videotapes were reviewed at the investigators' discretion.

Statistical analysis

A significance threshold of $P < .05$ was used for all analyses. Clopper-Pearson (exact) 95% confidence intervals (CIs) were used for unadjusted estimates of proportions. Multivariate logistic regression, augmented by generalized estimating equations, provided hypothesis tests as well as estimates of effects of interest, with adjustment for

TABLE 1. Demographic and endoscopy data

	Total no. of patients	Male	Female	Mean age, Y
Sex/age, y	130	82 (63.1%)	48 (36.9%)	57
Endoscopic findings				
Barrett's esophagus length, cm	Mean, 1.82	IQR, 0-2		Total range, 0-15
Hiatal hernia size, cm	Mean, 1.59	Median, 1		
	Minnesota Gastroenterology, PA, cases		**Outside physician cases**	
Previous endoscopy, no. (%)	114 (87.7%)		16 (12.3%)	

IQR, interquartile range.

correlation within previous endoscopists. The Fisher exact test was used for categorical variables.

Institutional review board

The study was approved by the Institutional Review Board of Allina Health (Minneapolis, Minn), which was allowed access to all study-related source data/documents at any requested time. Informed consent was obtained from all patients before performing the surveillance endoscopy, and there was no follow-up other than usual care after the examination. The 3 investigators designed the study and collectively decided to submit the data for publication. The lead author wrote the first draft of the manuscript and the other investigators contributed to the subsequent revisions. All of the investigators vouch for the integrity of the data submitted.

RESULTS

Demographic and endoscopy data

By definition, all 130 study patients had previously received a diagnosis of BE. Eighty-two patients (63.1%) were male, and 48 (36.9%) were female, with a mean age of 57 years. All patients were receiving proton pump inhibitor (PPI) therapy, although the duration of PPI use was not assessed. There were 30 (23.1%) cases of previously diagnosed long-segment BE (>3 cm) and 100 (76.9%) cases of short-segment BE. Sixteen of 130 (12.3%) of the patients had their previous endoscopy performed by a non-Minnesota Gastroenterology physician, and the remainder had their examinations performed by a Minnesota Gastroenterology, PA, physician. Of the 130 cases, there were 51 different initial endoscopists from both inside and outside of Minnesota Gastroenterology, PA, and no initial endoscopist performed more than 4 endoscopies of the patients returning for the study (Table 1).

Follow-up expert examination

The total BE length of the cases ranged from 0 to 15 cm, with an interquartile range of 0 to 2 cm, and a mean length of 1.82 cm. Forty-six of 130 patients (35.4%) had no hiatal hernia and 100 of 130 patients (76.9%) had either no hiatal hernia or a hiatal hernia smaller than 3 cm. The mean hiatal hernia size was 1.59 cm, and the median hernia size was 1 cm. Forty-five of the 130 study procedures (34.6%) were performed by the first investigator (R.A.G.), 36 of 130 (27.7%) were performed by the second (J.I.A.), and 49 of 130 (37.7%) by the third (S.L.) (Table 1).

TABLE 2. Total revised and nonrevised BE diagnoses

Total no. of cases	130
Revised	42 (32.3%)
Nonrevised	88 (67.7%)

BE, Barrett's esophagus.
95% confidence interval, 24.4%-41.1% (Clopper-Pearson exact confidence interval).

Revision of diagnosis

Of the 130 patients who previously received a diagnosis of BE, 88 (67.7%) were confirmed to have BE with positive endoscopic and histologic findings, and 42 (32.3%) had their diagnosis revised to no BE (95% CI, 24.4%-41.1%) (Table 2). Of the 42 patients with revision of their diagnosis, 5 of 42 (11.9%) had no visible columnar-lined esophagus above the gastric folds but did have cardia intestinal metaplasia (Fig. 1, Table 3), and 18 of 42 patients (42.9%) had neither visible columnar-lined epithelium in the esophagus nor intestinal metaplasia (Fig. 2, Table 3). Nineteen of 42 patients with a revised diagnosis (45.2%) did appear to be visually consistent with BE, but intestinal metaplasia could not be confirmed on histology (Fig. 3, Table 3).

All 3 endoscopists agreed on all confirmed cases of BE and agreed on 38 of 42 of those cases with a revised diagnosis. There was no statistical difference in revision rates among the 3 study endoscopists when adjusted by baseline covariates (for each of the 2 pairwise comparisons: $P = .170$

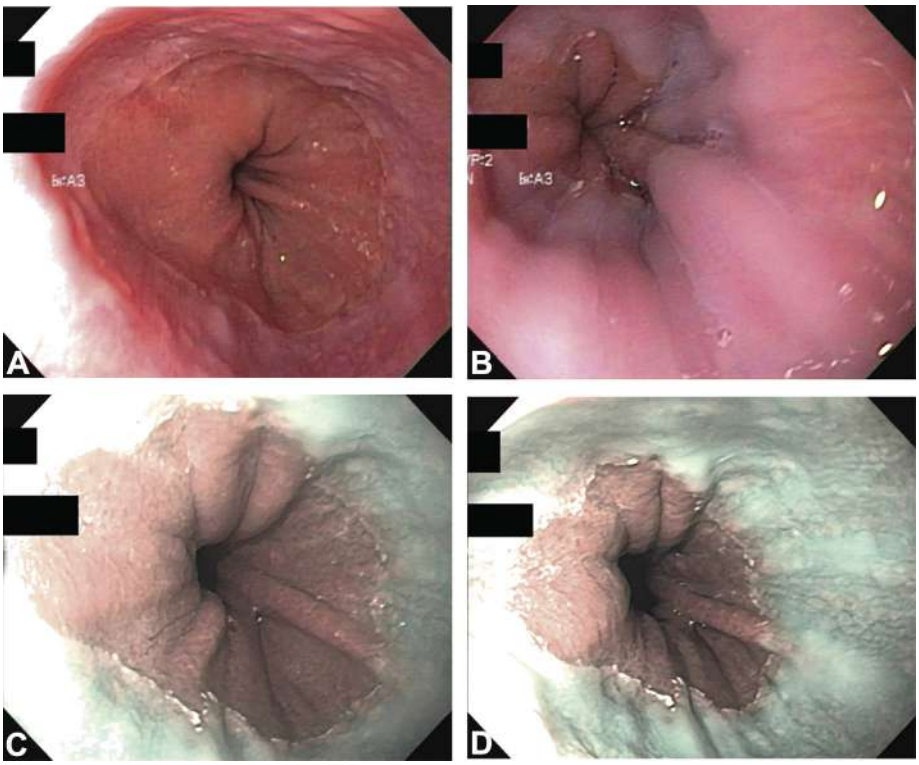

Figure 1. Example of a patient who initially received a diagnosis of Barrett's esophagus that was revised to cardia intestinal metaplasia. There was no visible columnar epithelium in the tubular esophagus; biopsies of the cardia region demonstrated goblet cells. The top photos are during white light endoscopy and the bottom photos used narrow-band imaging. Photos on the left show the gastroesophageal junction expanded with air; the air was suctioned and the gastroesophageal junction collapsed, in the photos on the right.

and $P = .322$), with the revision rates varying between 24.5% and 40.0%. In those patients with short-segment BE, 41 of 100 (41%) had their diagnosis revised, but only 1 of 30 (3.3%) patients with long-segment BE had the initial diagnosis revised ($P = .003$, Fisher exact test).

The site of the initial diagnostic endoscopy was also a predictor of revision of diagnosis. Nine of the 16 patients (56.3%) initially diagnosed with BE "outside" of Minnesota Gastroenterology, PA (i.e. initial procedure performed by a non-Minnesota Gastroenterology, PA, physician), were revised, whereas 33 of 114 (28.9%) Minnesota Gastroenterology, PA, cases were revised ($P = .044$). Additional predictors of revision of diagnosis included younger age ($P = .002$), female sex ($P = .011$), shorter BE length ($P = .003$), and shorter length or absence of hiatal hernia ($P = .007$ and .030, respectively). There were no cases consistent with *H pylori*–induced intestinal metaplasia, ie, none of the patients demonstrated intestinal metaplasia of the body or antrum (Table 4).

TABLE 3. 42 revised cases

No. of cases	Visible columnar epithelium	Intestinal metaplasia
5	–	+ (cardia)
18	–	–
19	+	–

CONCLUSIONS

In theory, there is no difference between theory and practice, but in practice there is.

Jan L. A. van de Snepscheut

In this study, on expert review, 42 of 130 patients (32.3%) (95% CI, 24.4%-41.1%) receiving a diagnosis of BE based on a previous endoscopy, did not have the original diagnosis confirmed, herein termed revision of BE. To the extent that this sample is representative of general practice in the United States, based on the CI, at least 24.4% of patients currently diagnosed with BE in the United States would not have had the diagnosis verified with a careful repeat endoscopic examination and repeat surveillance biopsies. The strengths of this study include the wide sampling of cases, with patients drawn from 51 endoscopists in the Minneapolis/St. Paul, Minnesota area, the intensive prestudy endoscopic preparation and consensus definitions, and the expertise of the endoscopists and pathologists. With focused and consensus review of certain

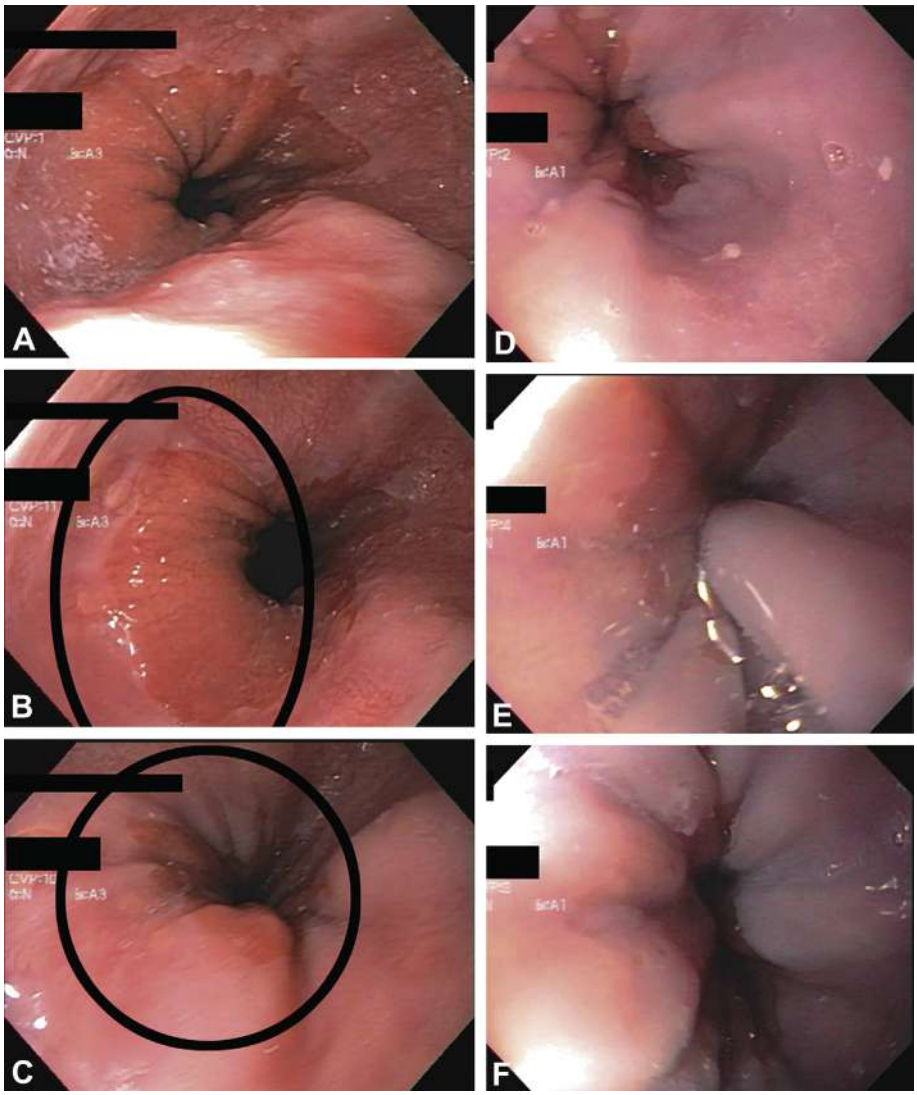

Figure 2. Compilation of photos from 2 patients (**A-C, D-F**) initially diagnosed with Barrett's esophagus and revised to normal. At the study endoscopy there was no visible esophageal columnar epithelium and there were no goblet cells seen in any biopsy specimen. Biopsy samples were taken at and just distal to the z-line in four quadrants. **B,** Demonstrates (black oval) the GE junction in the first patient expanded with air. **C,** The same region (black circle) is seen when the air was suctioned and the GE junction collapsed.

aspects of upper endoscopy, in particular, the region of the GEJ, the revision rates were consistent across 3 different endoscopists. Weaknesses of the study include the retrospectively identified cohort and the unbalanced study design across covariates (eg, outside cases, sex, hernia, BE length, age). Also, the initial pathology specimens were not re-read by expert pathologists; we relied on the pathology reports. The study design also precludes us from examining to what degree the underdiagnosis of BE might occur.

The association between previous BE length and overdiagnosis was examined, and virtually all of the diagnosis reversals in this study occurred in patients with previously "termed" short-segment BE; indeed in 23 of 42 revisions (54.8%), we could not identify any columnar-lined epithelium proximal to the gastric folds. As previously noted, this suggests limitations of the endoscopic diagnosis of BE and a high-degree of misinterpretation of columnar-lined epithelium in the region of the GEJ. In this study, the ratio of short-segment to long-segment BE was more than 3:1, which is greater than that reported in some publications. Additional predictors of diagnosis reversal included younger age, female sex, and small or absent hiatal hernia, again suggesting overzealous interpretation of the GEJ in patients with a low epidemiologic likelihood of having BE.

In 23 of 42 patients with diagnosis revision with no visible columnar lined esophagus in the distal tubular esophagus, 5 had cardia intestinal metaplasia and 18 were negative for cardia intestinal metaplasia (ie, no goblet cells). In 37 of 42 patients with diagnosis revision, we could not confirm any intestinal metaplasia in either the region of the GE junction or the 19 patients with

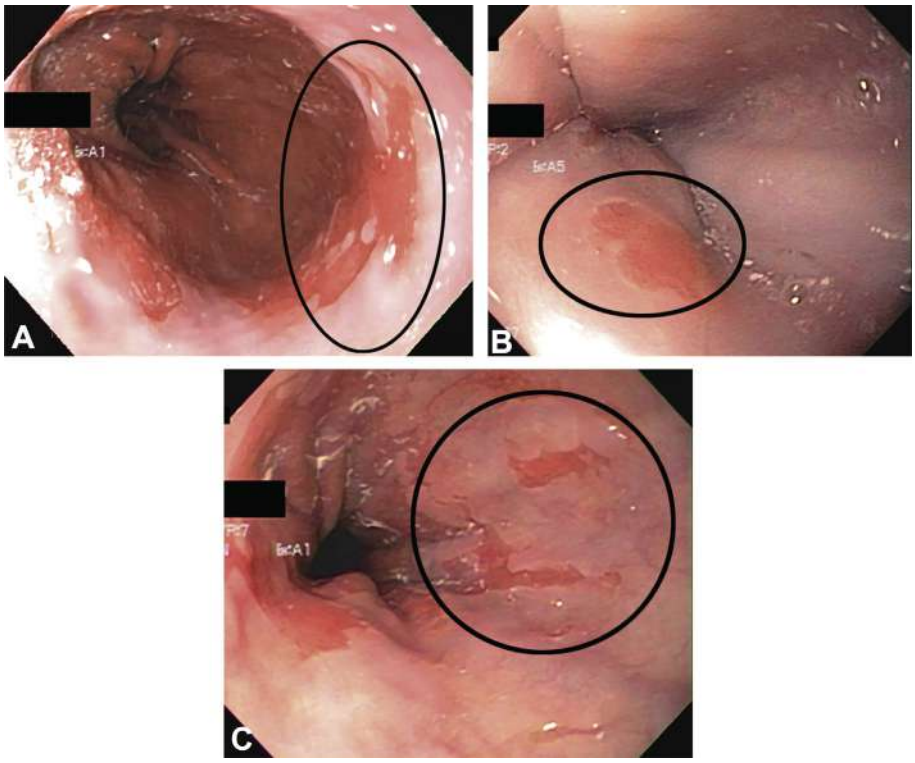

Figure 3. A, B, C, Compilation of photos from 3 patients with visible columnar epithelium in the tubular esophagus, but negative for goblet cells when biopsied during the study exam. The black circles in **A, B,** and **C** denote areas of specific biopsy.

columnar-lined epithelium extending proximal to the gastric folds. Why was the original intestinal metaplasia histology not confirmed on the study examination? We used standard-size forceps and an every 2-cm biopsy protocol for the study examinations, and this may have contributed to an intestinal metaplasia "miss rate" attributable to sampling error; however, we used the same biopsy protocol as that used by the majority of original endoscopists in which the intestinal metaplasia was initially found. Additionally, it is not clear from the GI literature whether jumbo forceps or every 1-cm biopsies make a significant difference in the "find" rate because these practices have never been prospectively compared.[1,16,17] Also, the recent AGAI BE consensus document sanctions every 2-cm 4-quadrant biopsies and is agnostic on standard size versus jumbo forceps.[1]

PPI-induced squamous overgrowth may well have occurred in some of the patients, obscuring subepithelial BE, but all of these patients were receiving PPI therapy, many long term, and it is entirely possible that any squamous overgrowth would have preceded the initial diagnostic examination, not just the study examination. In addition, it is unclear in the GI literature whether standard-size forceps obtain deep enough specimens to consistently identify this issue.[18-20]

How should we classify patients with columnar-lined epithelium in the distal tubular esophagus initially positive for intestinal metaplasia, but not confirmed on a follow-up examination? Should these patients be placed in the same bucket with those patients with true BE confirmed on follow-up examinations? Per protocol, we reversed the diagnosis in this study, but others might prefer additional confirmatory negative examinations. What should one do with these patients? Continue surveillance? If so, at what interval? Or should we end surveillance? If we still continue to label these patients as having BE based on a previous examination, consider the burdensome cost of this diagnosis to the individual and society, insurability issues, and patient psychological stress. These issues, however, are beyond the scope of this article.

Given the accepted limitations and lack of uniform consensus of endoscopic definitions in BE diagnosis, it is surprising that there has not been more consideration of the impact of incorrect, overdiagnosis of BE. Indeed published, large, retrospective epidemiologic series of BE prevalence and cancer progression risk accept previous endoscopic reports at face value and discount the possibilities of inadvertent cardia intestinal metaplasia or the lack of confirmation of diagnosis on repeat examination. Many of these large series do not even require a minimum BE length for study exclusion.[12,13,21]

In a multicenter Veterans Affairs study, using definitions similar to ours, Kim et al[22] performed repeat endoscopy in 82 patients 6 weeks after an initial diagnosis of BE and found that they could not confirm the diagnosis in 18% of those with long-segment BE (>3 cm), because

TABLE 4. Revised cases by variables

Age and sex			
	Total cases, no.	**Revised, no. (%)**	**Nonrevised, no. (%)**
Male	82	21 (25.6)	61 (74.4)
Female	48	21 (43.8)	27 (56.3)
Mean age, y	57	52	59
(Mean age, $P < .002$; sex, $P < .011$)			
Total Barrett's length (range, 1-15 cm)			
	Total cases	Revised	Nonrevised
Average	1.82	0.21	2.58
($P = .003$)			
Previous long- vs short-segment Barrett's			
	Total cases, no.	Revised, no. (%)	Nonrevised, no. (%)
Long	30	1 (3.3)	29 (96.7)
Short	100	41 (41.0)	59 (59.0)
($P = .003$)			
Mean hiatal hernia size, cm			
	Total cases	Revised	Nonrevised
Average	1.59	0.60	2.06
($P = .007$)			
Site of previous EGD			
	Total cases, no.	Revised, no. (%)	Nonrevised, no. (%)
MN Gastro	114	33 (28.9)	81 (71.1)
Outside	16	9 (56.3)	7 (43.8)
51 unique initial providers ($P = .044$)			
Cases performed per provider			
	Total cases, no.	Revised, no. (%)	Nonrevised, no. (%)
Allen	36	12 (33.3)	24 (66.7)
Ganz	45	18 (40.0)	27 (60.0)
Leon	49	12 (24.5)	37 (75.5)
No difference among investigators (adjusted for baseline covariates; $P = .170$, $P = .322$)			

MN Gastro, Minnesota Gastroenterology, PA.

of either discrepant histologic (unconfirmed intestinal metaplasia) or endoscopic findings and, similar to our results, in 9 of 27 (33%) of those with short-segment BE. Of the 9 patients with unconfirmed short-segment BE, 4 of 9 had columnar-lined epithelium but were negative for histologic goblet cells at follow-up endoscopy. In a European multicenter study, Meining et al[23] repeated endoscopy 30 months after the initial BE diagnosis and in a subgroup with initial visible esophageal columnar-lined epithelium with goblet cells was unable to reproduce a BE diagnosis in approximately 30%. In this study, histology was confirmed in 4 of 4 patients with long-segment BE; however, 5 of 12 patients with short-segment BE were negative for goblet cells at follow-up. Our study confirms these observations in a large cohort of patients, assessing the initial endoscopic diagnosis of multiple gastroenterologists in a standard American practice, ambulatory endoscopy setting.

In conclusion, we identified a large number of patients previously considered to have BE in whom the diagnosis could not be confirmed, with important ramifications for increased costs, reduced insurability, and psychological stress because of the perceived cancer risk.[24] The implications of this study suggest the need for a better definition of the GEJ, stricter accountability for a BE diagnosis, and improved education of endoscopists. In addition, published estimates of the prevalence of BE and cancer risk based on retrospective chart reviews of unconfirmed endoscopic diagnoses of BE, particularly those studies with a large number of patients with short-segment BE or in which the accepted diagnostic BE length is not predefined, may need to be reconsidered.[12]

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