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Racial Disparity in Sex Distribution, Prevalence and Incidence of Dysplasia in Barrett's Esophagus

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Abstract

Goals—Our aim was to study the prevalence of dysplasia and progression to high-grade dysplasia (HGD) and esophageal adenocarcinoma (EAC) in African Americans (AA) with Barrett's esophagus (BE) and compare to Non-Hispanic White (NHW) controls.

Background—BE, a precursor of EAC, is a disease of predominantly white men and is uncommon in AA. The prevalence of dysplasia and progression to HGD and EAC in AA with BE is not clearly known.

Study—All AA or NHW patients with confirmed BE i.e. specialized intestinal metaplasia seen between 2002 and 2013 at our institution were included. Variables such as age, gender, medication use, body mass index, date of endoscopy, hiatal hernia size, BE length, and histological findings were noted. Progression to HGD/EAC was evaluated.

Results—Fifty two AA and 2394 NHW patients with BE were identified. There was a higher percentage of women in AA cohort (46.2%) than NHW cohort (24.9% $p < 0.001$). Nondysplastic BE was more prevalent in AA than in NHW (80.8% vs. 68.4%, $p = 0.058$). In the surveillance cohort of 20 AA and 991 NHW, no racial differences in progression to HGD/EAC were observed during a median follow-up of 43 months.

Conclusions—This study includes the largest number of AA with histologically confirmed BE reported so far. 46.2 % of AA with BE in our study were women. There was a trend towards higher prevalence of nondysplastic BE in AA compared to NHW.

Keywords

esophageal cancer; carcinogenesis; dysplasia; patient management

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INTRODUCTION

Esophageal adenocarcinoma (EAC) and its precursor, Barrett's esophagus (BE) are predominantly diseases of white men^{1, 2}. BE is relatively uncommon in African Americans (AA), and thus, sparse data exist regarding the incidence and prevalence rates of BE and BE-related dysplasia in AA³⁻⁵. However, recent studies report an increasing incidence of EAC in AA⁶. Although there is increase in incidence of EAC in NHW and AA over time, the racial disparities have not changed. Analysis of Surveillance, Epidemiology and End Results data from 1983 to 2012 show incidence rate of EAC of 0.5 per 100,000 person-years in AA compared to 2.4 in whites. Also, over the three decades, male to female ratio in AA with EAC have decreased from 4.81 in 1983 to 1992 to 3.33 from 2003 to 2012⁷.

There do not seem to be any racial differences in the prevalence of gastroesophageal reflux disease (GERD) related symptoms, but AA have lower prevalence of reflux esophagitis compared to non-Hispanic whites (NHW)^{3,8}. Although substantial evidence exists to support lower prevalence of BE in AA, a few studies reported no significant racial differences in BE. In a US study of 4,457 patients who underwent upper endoscopy, no significant racial differences were observed in the prevalence of BE, irrespective of GERD symptoms⁹. In another study based on veteran population, race was not a predictor for presence of BE¹⁰.

In addition, the prevalence of dysplasia in AA patients with BE also seems to be lower compared to NHW patients with BE. Khoury et al performed a single-center, retrospective analysis of 16 AA BE cases and 95 NHW BE cases, reporting that AA were less likely to have dysplastic BE (0% versus 7%)¹¹. In a recent study of 35 AA patients with BE, none of the patients had dysplasia as compared to 5.6% in NHW ($p=0.06$)⁴.

Once in a surveillance program, it is unknown if AA with BE are at same risk for progression to dysplasia/EAC as NHW patients. There are limited data about the disease course as well as the incidence of dysplasia and EAC in AA with BE^{12,13}. Hence, our aim was to 1) estimate the prevalence of dysplasia in AA patients with BE compared to NHW with BE and 2) assess the influence of race in progression to high-grade dysplasia (HGD) and EAC in BE. We hypothesized that there is a lower prevalence of dysplasia and EAC in AA patients with BE compared to NHW counterparts and that AA with BE are at a lower risk of developing HGD/EAC under surveillance.

MATERIALS AND METHODS

Study Population

Following approval by institutional review board, the patient population for the study was obtained from the prospectively collected Barrett's registry at Cleveland Clinic. This database includes BE patients seen in the department of Gastroenterology since 1979. In this study, all AA and NHW patients with BE that were seen between January 2002 and December 2013 were included. Patients of unknown race and race other than NHW or AA were excluded. Age, gender, medication such as proton pump inhibitors (PPI), histamine

receptor antagonists (H2RA), aspirin and nonsteroidal anti-inflammatory drugs (NSAID) use, body mass index (BMI), date of endoscopy, length of BE segment, size of hiatal hernia and histological findings were reviewed.

Patients were considered to have BE if there is salmon colored mucosa of any length on endoscopy with intestinal metaplasia was present on biopsy. The distance from the proximal end of the gastric folds to the squamocolumnar junction was calculated as the BE length. The biopsy results were read as no dysplasia, low-grade dysplasia (LGD), indefinite for dysplasia (IND), HGD and EAC as per Vienna classification¹⁴. IND was included under LGD as they share similar clinical management. The surveillance intervals varied depending on the presence of dysplasia and as per existing guidelines for BE surveillance¹⁵. For nondysplastic BE, endoscopy was repeated in one year to confirm absence of dysplasia and every 3 years thereafter. Since 2011, surveillance interval was extended to 3–5 years. For LGD, surveillance biopsy protocol consisted of four quadrant biopsies every 1–2 cm of BE with a standard or jumbo biopsy forceps every 6–12 months. Patients with HGD were referred for esophagectomy or endoscopic eradication therapy depending on year of diagnosis and their surgical candidacy. In most cases of dysplasia or EAC, diagnosis was confirmed by a gastrointestinal pathologist or reviewed at a consensus conference. Progression to LGD, HGD or EAC was evaluated during the follow-up. Follow-up time was defined as months from first endoscopy to progression or to last endoscopy if there was no progression. Patients who had dysplasia or EAC at initial endoscopy or within one year were considered prevalence cases and patients who developed LGD/HGD/EAC after at least one year follow-up were considered as incident cases.

Statistical Analysis

Data were presented as mean $\pm$ standard deviation, median [25th, 75th percentiles] or frequency (percent). To assess the differences between the groups, a univariable analysis was performed. Analysis of variance (ANOVA) or the non-parametric Kruskal-Wallis tests were used for continuous factors and Pearson's chi-square test or Fisher's Exact tests were used for categorical variables. A time-to-event analysis was performed to assess association between race and progression in the subgroup patients without HGD or EAC at baseline. Follow-up was truncated at 3 years since 50% of subjects were followed for less than 3 years after baseline EGD. Kaplan-Meier plots were constructed and log-rank tests were used to compare groups. A $p < 0.05$ was considered statistically significant. SAS version 9.4 (The SAS Institute, Cary, NC) was used to perform all analysis.

RESULTS

The total study population consisted of 2544 patients (Figure 1). The racial distribution was: NHW 2394 (94%), AA 52 (2%), Hispanic 18 (0.70%), Asian 4 (0.15%), American Indian 4 (0.15%), multiracial 5 (0.19%) and unknown/others 67 (2.63%). A total of 2446 were included in our analysis. The average age was 60 ± 13 years and 75% were men. Table 1 presents a description of patient characteristics. In comparison to NHWs, AA with BE were more likely to be women, and less likely to use PPIs. There was a trend towards higher

prevalence of nondysplastic BE in AA compared to NHW with 42 (80.8%) in AA versus 1638(68.4%) in NHW ($p=0.058$).

Among 2446 patients, 501 had HGD/EAC on first endoscopy and 42 developed HGD/EAC within one year. 892 patients did not have any surveillance information available. Therefore a total of 1011 patients (20 AA and 991 NHW) constituted the surveillance cohort and were included in the progression analysis (Table 2). Median follow-up time was 43 months [range: 21 – 81]. During this time, 19% of patients had progression of dysplasia. 134 NHW and 2 AA who had no dysplasia progressed to LGD; 20 NHW progressed from no dysplasia to HGD; 15 NHW and 1 AA with no dysplasia to cancer; 16 NHW from LGD to HGD; 3 NHW from LGD to cancer (Figure 2). There was no evidence to suggest that race was associated with progression to dysplasia in either patients without dysplasia or LGD at baseline (Figure 3). Because of the low number of AA that progressed ($n=3$) multivariable analysis stratified by baseline dysplasia could not be performed. After adjusting for gender for any progression in all the patients, race remained non-significant.

Twelve out of 20 (60 %) were women in AA surveillance cohort where as 275/991 (27.7%) were women in NHW cohort. There were no significant differences in age, BMI, BE segment length, hiatal hernia size, medication use or progression to HGD/EAC between AA and NHW surveillance cohorts (Table 2).

DISCUSSION

As per US Census Bureau, African Americans comprise of 12.6% of total population of United States and 10.4% of total population in Midwest¹⁶. In our Barrett's registry, only 5% of patients were AA suggesting a low prevalence of Barrett's in this population. Since 1980's, numerous studies have reported the preponderance of BE with EAC in NHW men and rarity in AA^{4,11,17,18}. Whether this is due to genetic differences leading to a protective phenotype or racial differences in the risk factors for BE is not entirely clear. The genetic factors to account for this racial disparity are focus of intense investigation but none have been identified so far. A factor that is worth bearing scrutiny is the higher prevalence of *H.pylori* infection in blacks as compared to NHW. In a US study of 7465 adults, age-adjusted prevalence for *H.pylori* was substantially higher among AA (52.7%) than among NHW (26.2%)¹⁹. Chronic gastritis from *H.pylori* leads to gastric atrophy and the subsequent reduced gastric acid production translates into a lower risk of BE. Supporting evidence comes from a recent meta-analysis which showed that *H.pylori* infection leads to a reduced risk of BE (relative risk 0.46; 95% CI, 0.40–0.53)²⁰. A recent study based on VA population highlighted the racial differences in risk factors for BE. However in this study, *H.pylori* negativity and high waist hip ratio was predictive of BE in NHW but not in AA. In AA, the risk factors for BE included a hiatus hernia ≥ 3 cm (OR 4.12; 95% CI, 1.57–10.81) and a history of GERD or PPI use (OR, 3.70; 95% CI, 1.40–9.78), whereas in NHW, high waist hip ratio (OR, 2.82; 95% CI, 1.41–5.63), hiatus hernia ≥ 3 cm (OR, 4.95; 95% CI, 3.05–8.03), PPI use (OR, 1.88; 95% CI, 1.33–2.66), and *H.pylori* (OR, 0.64; 95% CI, 0.41–0.99) were associated with BE risk⁴.

A significant finding of our study is attenuation of gender disparity in AA compared to NHW. In spite of the genetic protection conferred by the race, AA women were 46 % of total AA patients with BE compared to 25% of NHW women. In other words, female to male ratio was 1: 1.13 in AA compared to 1:3 in NHW. These findings support a joint gender and race interaction in the development of BE. One explanation for these findings could be a higher genetic load of susceptibility alleles in women in the admixed AA population. Prior studies of BE in AA reported small numbers of patients, hence meaningful interpretation regarding gender differences could not be done ^{4,21,22}.

Another explanation for this finding in our study may be racial and gender differences in BMI or adipokines. AA women have the highest rates of obesity in the U.S. -nearly 59 percent, compared to 33 percent in NHW women ^{23,24}. Increased BMI appears to be a risk factor for BE in women ¹⁹. As AA women are known to have an increased BMI in comparison to NHW counterparts ^{25,26}, it is conceivable that AA women are more predisposed to BE than NHW women. In our study, there were no significant differences in BMI, which raises the possibility of adipokines. Adipokines such as adiponectin and leptin may influence the association between visceral obesity and BE. High levels of leptin are associated with a two-fold increased risk of BE ²⁷ whereas adiponectin seems to have a protective effect at least in some studies ²⁸. Leptin levels are known to be higher in women than in men after adjusting for total body fat ²⁹. Among women, racial differences persist in the adipokine levels. AA women have lower total adiponectin (β : -3.40; 95% CI: -4.29, -2.52; $P < 0.001$) and higher leptin levels (β : 3.26; 95% CI: 1.36, 5.16; $P < 0.001$) compared to NHW women ³⁰. These factors may account for the gender attenuation we noted in AA women with BE.

In our study, the prevalence of nondysplastic BE was 68.4 % in NHW compared to 80.8% in AA group. This is in accordance with previous studies which showed lower prevalence of dysplasia in AA with BE. In a study of 95 NHW and 16 AA with BE, AAs were less likely to have dysplasia (0% versus 7%,) and less frequent long segment BE (12% versus 26%) ¹¹. In another study of 35 AA with BE, none of them had dysplasia as compared to 251 NHW patients with BE (236 no dysplasia, 13 LGD and 2 HGD $p = 0.06$) ⁴. Similarly, the incidence rates of EAC are also lower in AA compared to NHW. In a large veterans cohort with BE ($n = 29,536$), the incidence rate of EAC in AA was 2.62 per 1000 person-years compared to 3.34 in NHW with an incidence rate ratio of 0.78 suggesting lower risk of progression ¹³. In our study, we did not find any racial differences in progression to HGD/EAC possibly due to small numbers and short duration of follow up.

The main strength of this study is that it includes a large number of AA with confirmed BE i.e. intestinal metaplasia. Surveillance information was available which allowed us to assess for risk of progression to HGD/EAC. Most of the cases of suspected dysplasia or cancer were confirmed by a gastrointestinal pathologist or reviewed at a consensus conference thereby reducing the inter-observer variability.

Our study has several limitations. Since this was not a population-based study, there may be referral bias. Such a bias alone, however, would not explain the large racial differences found in our study. BMI values were missing for several patients which may impact the

study findings. Nonetheless, it is the visceral adiposity or waist hip ratio which is associated with risk of BE than BMI per se. The other limitation is the lack of information about *H.pylori* status in our study population. Finally, the finding of higher proportion of AA women with BE may be due to gender differences in the health seeking behavior among AA. AA women are significantly more likely to seek professional help and utilize physician services than AA men ³¹.

In conclusion, male to female gender ratio is attenuated in AA with BE compared to their NHW counterparts. There is lower prevalence of BE associated neoplasia in AA compared to NHW. The genetic factors and environmental factors contributing to this disparity require further study.

Abbreviations

AA	African Americans
BE	Barrett's Esophagus
BMI	Body Mass Index
EAC	Esophageal Adenocarcinoma
H. Pylori	Helicobacter pylori
HGD	High-Grade Dysplasia
H2RA	histamine receptor antagonists
LGD	Low-Grade Dysplasia
NHW	Non-hispanic whites
NSAID	nonsteroidal anti-inflammatory drugs
PPI	proton pump inhibitors

References

1. A Snapshot of Esophageal Cancer. National Cancer Institute; Nov 5, 2014. Available at: <http://www.cancer.gov/research/progress/snapshots/esophageal>
2. Siegel RL, Miller KD, Jemal A. Cancer statistics. 2015; 65:5–29.
3. El-Serag HB, Petersen NJ, Carter J, et al. Gastroesophageal reflux among different racial groups in the United States. Gastroenterology. 2004; 126(7):1692–9. [PubMed: 15188164]
4. Nguyen TH, Thrift AP, Ramsey D, et al. Risk factors for Barrett's esophagus compared between African Americans and non-Hispanic Whites. Am J of Gastroenterol. 2014; 109(12):1870–80. [PubMed: 25420546]
5. Yousef F, Cardwell C, Cantwell MM, et al. The incidence of esophageal cancer and high-grade dysplasia in Barrett's esophagus: a systematic review and meta-analysis. Am J Epidemiol. 2008; 168(3):237–49. [PubMed: 18550563]
6. Cook MB, Chow WH, Devesa SS. Oesophageal cancer incidence in the United States by race, sex, and histologic type, 1977–2005. Br J Cancer. 2009; 101:855–9. 7. [PubMed: 19672254]

7. Thrift AP, El-Serag HB. Sex and Racial Disparity in Incidence of Esophageal Adenocarcinoma: Observations and Explanations. *Clin Gastroenterol Hepatol*. 2016; 14(3):330–2. [PubMed: 26528803]
8. Vega KJ, Chisholm S, Jamal MM. Comparison of reflux esophagitis and its complications between African Americans and non-Hispanic whites. *World J Gastroenterol*. 2009; 15(23):2878–81. [PubMed: 19533809]
9. Fan X, Snyder N. Prevalence of Barrett's esophagus in patients with or without GERD symptoms: role of race, age, and gender. *Dig Dis Sci*. 2009; 54:572–7. [PubMed: 18654849]
10. Eloubeidi MA, Provenzale D. Clinical and demographic predictors of Barrett's esophagus among patients with gastroesophageal reflux disease: a multivariable analysis in veterans. *J Clin Gastroenterol*. 2001; 33(4):306–9. [PubMed: 11588545]
11. Khoury JE, Chisholm S, Jamal MM, et al. African Americans with Barrett's esophagus are less likely to have dysplasia at biopsy. *Dig Dis Sci*. 2012; 57(2):419–23. [PubMed: 21909989]
12. Corley DA, Kubo A, Levin TR. Race, ethnicity, sex and temporal differences in Barrett's oesophagus diagnosis: a large community-based study, 1994–2006. *Gut*. 2009; 58(2):182–188. [PubMed: 18978173]
13. Shakhathreh MH, Duan Z, Kramer J, et al. The incidence of esophageal adenocarcinoma in a national veterans cohort with Barrett's esophagus. *Am J Gastroenterol*. 2014; 109(12):1862–8. [PubMed: 25331350]
14. Schlemper RJ, Riddell RH, Kato Y, et al. The Vienna classification of gastrointestinal epithelial neoplasia. *Gut*. 2000; 47:251–255. [PubMed: 10896917]
15. Spechler SJ, Sharma P, et al. American Gastroenterological Association medical position statement on the management of Barrett's esophagus. *Gastroenterology*. 2011; 140(3):1084–91. [PubMed: 21376940]
16. 2010 Source: U.S. Census Bureau, 2010 Census Redistricting Data (Public Law 94–171) Summary File, Table P1
17. Smith RL, Hamilton SR, Boitnott JK, et al. The spectrum of carcinoma arising in Barrett's Esophagus. *Am J Surg Pathol*. 1984;8:56–73.
18. Rogers EL, Goldkind SF, Iseri OA, et al. Adenocarcinoma of the Lower Esophagus: A Disease Primarily of White Men with Barrett's Esophagus. *J Clin Gastroenterol*. 1986; 8(6):613–618. [PubMed: 3805655]
19. Logan RPH, Walker MM. Epidemiology and diagnosis of *Helicobacter pylori* infection. *BMJ*. 2001; 323:920. [PubMed: 11668141]
20. Fischbach LA, Nordenstedt H, Kramer JR, et al. The association between Barrett's esophagus and *Helicobacter pylori* infection: a meta-analysis. *Helicobacter*. 2012; 17:163–75. [PubMed: 22515353]
21. Abrams JA, Fields S, Lightdale CJ, et al. Racial and ethnic disparities in the prevalence of Barrett's esophagus among patients who undergo upper endoscopy. *Clin Gastroenterol Hepatol*. 2008; 6:30–4. [PubMed: 18063419]
22. Wang A, Mattek NC, Holub JL, et al. Prevalence of complicated gastroesophageal reflux disease and Barrett's esophagus among racial groups in a multi-center consortium. *Dig Dis Sci*. 2009; 54(5):964–71. [PubMed: 19255852]
23. Flegal KM, Carroll MD, Kit BK, et al. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999–2010. *JAMA*. 2012; 307:491–7. [PubMed: 22253363]
24. Jacobson BC, Chan AT, Giovannucci EL, et al. Body mass index and Barrett's oesophagus in women. *Gut*. 2009; 58(11):1460–6. [PubMed: 19336423]
25. Evans EM, Rowe DA, Racette SB, et al. Is the current BMI obesity classification appropriate for black and white postmenopausal women? *Int J Obes*. 2006; 30(5):837–43.
26. Rahman M, Berenson AB. Accuracy of current body mass index obesity classification for white, black, and Hispanic reproductive-age women. *Obstet Gynecol*. 2010; 115(5):982–8. [PubMed: 20410772]
27. Chandar AK, Devanna S, Lu C, et al. Association of Serum Levels of Adipokines and Insulin With Risk of Barrett's Esophagus: A Systematic Review and Meta-Analysis. *Clin Gastroenterol Hepatol*. 2015; 13(13):2241–55. [PubMed: 26188139]

28. Greer KB, Falk GW, Bednarchik B, et al. Associations of Serum Adiponectin and Leptin With Barrett's Esophagus. *Clin Gastroenterol Hepatol*. 2015; 13(13):2265–72. [PubMed: 25737442]
29. Havel PJ, Kasim-Karakas S, Dubuc GR, et al. Gender differences in plasma leptin concentrations. *Nat Med*. 1996 Sep; 2(9):949–50. [PubMed: 8782440]
30. Khan UI, Wang D, Sowers MR, et al. Race–ethnic differences in adipokine levels: the Study of Women's Health Across the Nation (SWAN). *Metabolism*. 2012; 61(9):1261–9. [PubMed: 22444780]
31. Neighbors HW, Howard CS. Sex differences in professional help seeking among adult black Americans. *Am J Community Psychol*. 1987; 15(4):403–17. [PubMed: 3673952]

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Figure 1.
Study Population

Figure 2.
Progression to HGD/EAC in Surveillance Cohort. HGD= High-grade dysplasia, EAC= Esophageal adenocarcinoma

Figure 3. Progression to HGD/EAC in African-American and Non Hispanic Whites with Barrett's Esophagus. HGD= High-grade dysplasia, EAC= Esophageal adenocarcinoma

Table 1

Patient Characteristics-Prevalence Cohort

Factor	Total (N=2446)	NHW (N=2394)	African-American (N=52)	p-value
Age at BE diagnosis(years)	59.8±13.2	59.8±13.2	60.7±13.2	0.63 ^a
Male (%)	1825(74.6)	1797(75.1)	28(53.8)	<0.001 ^c
BMI (all)	29.7±6.1	29.7±6.0	30.2±7.4	0.62 ^a
BMI (men)	29.6±5.7	29.6±5.6	28.6±6.4	0.42 ^a
BMI (women)	30.1±7.2	30.0±7.1	31.8±8.2	0.27 ^a
BE Length (cm)	3.2±3.6	3.2±3.6	2.3±2.6	0.075 ^a
Hiatal Hernia (cm)	2.1±2.6	2.1±2.7	2.1±2.2	0.99 ^a
Aspirin	402(17.8)	395(17.9)	7(14.0)	0.47 ^c
NSAIDs	638(28.3)	628(28.5)	10(20.0)	0.19 ^c
PPI	788(35.0)	778(35.3)	10(20.0)	0.025 ^c
H2 RA	358(15.9)	350(15.9)	8(16.0)	0.98 ^c
Histology	2394	2394	52	0.23 ^c
. No dysplasia	1680(68.7)	1638(68.4)	42(80.8)	
. LGD	265(10.8)	260(10.9)	5(9.6)	
. HGD	218(8.9)	216(9.0)	2(3.8)	
. EAC	283(11.6)	280(11.7)	3(5.8)	

Values presented as Mean ± SD or N (column %).

p-values:

^a =ANOVA,^c =Pearson's chi-square test,^d =Fisher's Exact test.

BE= Barrett's Esophagus; BMI= Body Mass Index; EAC= Esophageal Adenocarcinoma; HGD= High-Grade Dysplasia; histamine receptor antagonists= H2RA; LGD= Low-Grade Dysplasia; NHW= Non-hispanic whites; nonsteroidal anti-inflammatory drugs = NSAID; proton pump inhibitors = PPI.

Table 2

Patient Characteristics-Surveillance Cohort

Factor	Total (N=1011)	NHW (N=991)	African-American (N=20)	p-value
	n	Summary	n	Summary
Age at BE Diagnosis (Years)	991	57.9±12.7	20	61.3±12.0
Male (%)	991	716(72.3)	20	8(40.0)
BMI (All)	510	29.7±5.9	16	30.4±6.3
BMI (Male)	365	29.5±5.4	6	29.7±5.7
BMI (Female)	145	29.8±6.8	10	30.7±6.9
BE Length (cm)	991	3.0±3.4	20	2.9±2.8
Hiatal Hernia (cm)	991	2.2±3.1	20	3.0±2.8
Aspirin	938	257(27.4)	20	3(15.0)
NSAIDs	938	385(40.2)	20	5(25.0)
PPI	938	457(48.7)	20	6(30.0)
H2 RA	938	273(29.1)	20	5(25.0)
Baseline Histology	991		20	
· No dysplasia	843(83.4)	826(83.4)		17(85.0)
· LGD	168(16.6)	165(16.6)		3(15.0)
Progression to HGD/EAC	991	54(5.4)	20	1(5.0)
Any Progression	991	188(19.0)	20	3(15.0)
Follow -up (months)	991	43.1[20.5,81.6]	20	36.9[29.4,49.7]

Values presented as Mean ± SD, Median [P25, P75] or N (column %).

p-values:

^a =ANOVA,^b =Kruskal-Wallis test,^c =Pearson's chi-square test,

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p = Fisher's Exact test.

BE = Barrett's Esophagus; BMI = Body Mass Index; EAC = Esophageal Adenocarcinoma; HGD = High-Grade Dysplasia; histamine receptor antagonists = H2RA; LGD = Low-Grade Dysplasia; NHW = Non-hispanic whites; nonsteroidal anti-inflammatory drugs = NSAID; proton pump inhibitors = PPI