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Title:
Prevalence and evolution of newly diagnosed autoimmune gastritis in a large Spanish retrospective cohort

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Prevalence and evolution of newly diagnosed autoimmune gastritis in a large Spanish retrospective cohort.

Study population

Single center retrospective study

↓

Positive Parietal cell antibodies
+
Patients with upper endoscopy
(2013-2023)

Methods

Prevalence of autoimmune gastritis

Endoscopic features

Histological stages:
- Stage 0: potential
- Stage I: non-atrophic gastritis
- Stage II: atrophic gastritis
- Stage III: dysplasia, NET or adenocarcinoma

Evolution at follow-up endoscopy

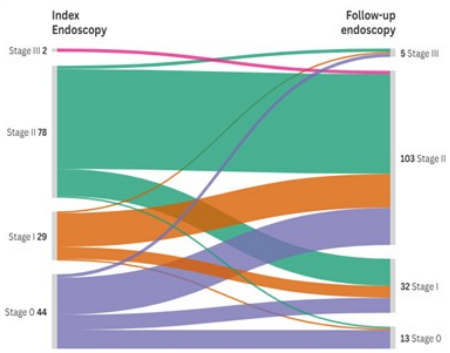
Outcomes

Prevalence: 1.6%
(95% CI 1.4%-1.7)

Histology
Stage 0: 105 patients (24.7%),
Stage I: 99 patients (23,2%)
Stage II: 215 patients (50.5%)
Stage III: 7 patients (1.6%)

↑ Evolution at follow-up endoscopy:
Δ 18.7% of advanced stages (II+III)

↓ Δ 20.2 % of Potential AIG



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Prevalence and evolution of newly diagnosed autoimmune gastritis in a large Spanish retrospective cohort

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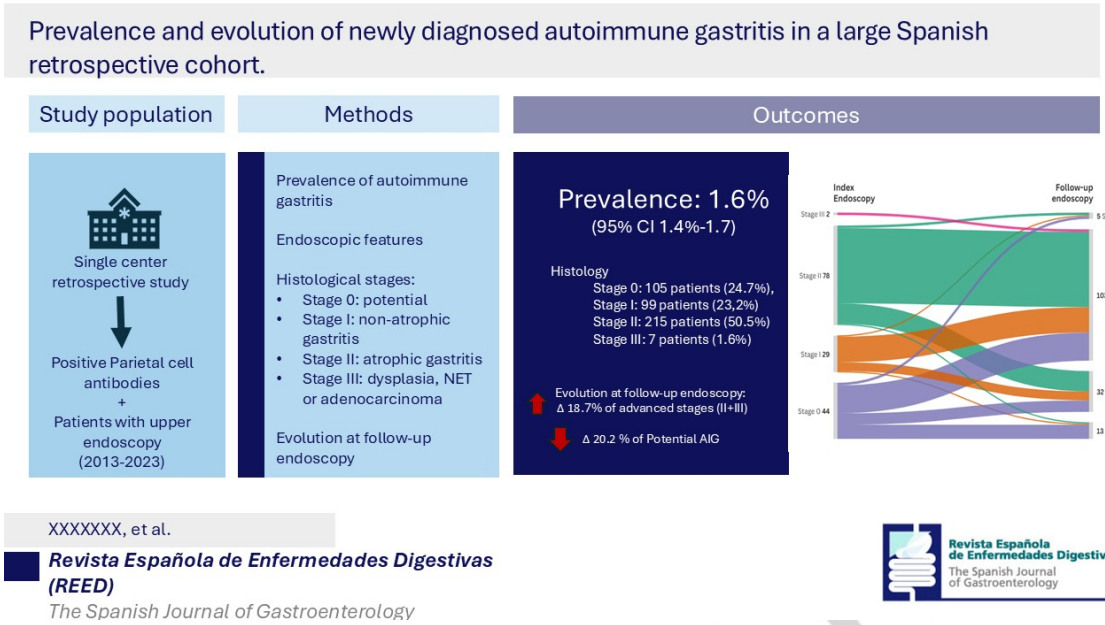

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Abstract:

Introduction: There is a growing interest in autoimmune gastritis (AIG), particularly regarding its prevalence and natural history.

Methods. Retrospective observational study including all patients with positive parietal cell antibodies and performance of gastroscopy between 2013 and 2023. The first subsequent gastroscopy was defined as follow-up endoscopy and considered for histological comparison. Categorization of histological stages was made into stage 0 (potential), stage I (non-atrophic gastritis), stage II (atrophic gastritis) and stage III (dysplasia, neuroendocrine tumor or adenocarcinoma).

Results: a total of 426 patients were included, 316 females with a median of 54.4 years (IQR 45.3-63.2). During this period, a total of 26798 patients underwent at least one upper endoscopy, so the prevalence of AIG was 1.6% (95% CI 1.4-1.7%). Histologically, 105 patients were classified as potential AIG (24.7%), 99 patients as stage I (23,2%), 215 patients as stage II (50.5%) and 7 patients as stage III (1.6%). 153 patients presented a follow endoscopy. A significant increase of advanced stages was observed at follow-up (difference 18.7% 95% CI 7.6%-29%; p=0.001) and a decrease of potential GAI (difference 20.2% 95% CI 11.8% -28.7%; p<0.001). At baseline 39 patients

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exhibited hyperplastic polyps (9.2%), 8 patients fundic gland polyps (1.9%), 3 adenomas with low-grade dysplasia (0.7%) and 3 patients presented G1 neuroendocrine tumours (0.7%). Only one patient (0.2%) was diagnosed with signet-ring cell gastric carcinoma.

Conclusions: AIG presented a low prevalence among patients undergoing gastroscopy. Biopsies in patients with positive parietal cell antibodies revealed that around half of the population exhibited significant atrophy and a notable progressive disease.

Keywords: Autoimmune gastritis. Neuroendocrine tumor. Gastric adenocarcinoma. Atrophy.

Introduction

Autoimmune gastritis (AIG) is a chronic condition caused by anti-parietal cell antibodies and/or anti-intrinsic factor production and gastric oxyntic mucosal damage. It is characterized by a corpus-dominant atrophy in contrast to helicobacter pylori infection-related gastritis [1] [2]. The diagnosis in Western countries is based on histopathology and serology. Although considered a preneoplastic condition, the risk of gastric adenocarcinoma has been questioned in some recent studies [3]. Nevertheless, it seems to be a progressive disease, even in patients initially without atrophy or signs of chronic gastritis at baseline defined as potential AIG could progress to more advanced stages [4].

In recent years, there has been a growing interest in gaining a deeper understanding of this condition, particularly concerning the role of Helicobacter pylori (HP) infection, the established diagnostic criteria, and the associated risks of gastric cancer progression [5].

Nevertheless, few studies have assessed the features of AIG in Spain [6]. Therefore, this study aimed to evaluate the prevalence, endoscopic and histological features of the newly diagnosed AIG and its evolution.

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Methods:

Study design.

A single-center retrospective observational study was conducted at Hospital Universitario Infanta Elena. The study was approved by the Institutional review board of Instituto de Investigación Fundación Jiménez Díaz (EO152-24_HIE). All authors had access to the complete dataset and have reviewed and approved the final manuscript.

Patients

Adult patients over (>18 years) with positive parietal cell antibodies (≤1/160) (PCA) who underwent a gastroscope within +/-1 year from the PCA determination from June 2013 to June 2023 with biopsies according to Sydney protocol were eligible for inclusion.

Exclusion criteria included the presence of Helicobacter infection in gastric biopsies, a positive Helicobacter antigen detected in faeces (<12 months), and patients who had undergone gastroscopy without biopsies or had incomplete biopsies. Additionally, any other endoscopic or histological indications of drug-related causes, bacterial, or gastric infection were considered exclusion criteria. Individuals who had undergone treatment for Helicobacter pylori up to 12 months before the index endoscopy were considered for inclusion.

Definitions

The index endoscopy was defined as the first endoscopy within a year of a positive PCA. Any subsequent gastroscopy with biopsies according to the Sydney protocol was considered as a follow-up gastroscope.

Histological classification was simplified into four stages based on previously published data according to histopathological features at oxyntic mucosa[7].

- Stage 0 (potential): positive PCA without any feature of chronic gastritis or atrophic mucosa.

- Stage 1 (non-atrophic gastritis): positive PCA and lymphocytic and plasmatic cell infiltration of the oxyntic gastric mucosa.
- Stage 2 (atrophic gastritis): positive PCA and lymphocytic and plasmatic cell infiltration and atrophy of the oxyntic gastric mucosa.
- Stage 3 (complicated): any of the above stage and the presence of enterocromaphine cell dysplasia, type 1 neuroendocrine tumors, intraepithelial glandular neoplasia or adenocarcinoma.

The presence of pseudopyloric or intestinal metaplastic changes was also recorded. Other polypoid lesions, including hyperplastic and fundic gland polyps, were also noted.

Data measurements

The primary outcome was the prevalence of newly diagnosed autoimmune gastritis (AIG) which was determined by considering all patients who underwent at least one gastroscopy throughout the study period. So, patients who underwent more than one endoscopy were considered as a single count in the data analysis. Secondary outcomes included the rate of GAI stages, endoscopic polypoid and non-polypoid findings, the prevalence of autoimmune comorbidities (autoimmune thyroiditis, Polyglandular syndrome, Sjögren's Syndrome, Rheumatoid arthritis, type I diabetes, autoimmune diabetes, primary biliary cholangitis, celiac's disease, inflammatory bowel diseases), associated gastrointestinal symptoms and evolution of AIG.

Dysplastic, pseudopyloric and intestinal metaplastic changes were recorded separately for the gastric antrum/incisura and body. Advanced AIG was considered whenever stage II or III were noted.

PCA were categorized according to dilutions into 1/160; 1/320; 1/640 and 1/1280.

Evolution of AIG.

The evolution of AIG was recorded whenever a patient underwent at least one follow-up gastroscopy, regardless of the reason for the repeated procedure. A minimum of a 1 -year interval between the index and follow-up endoscopy was required. For histological comparison, stages were categorized into advanced AIG whenever stages II or III were present.

Statistical analysis.

Continuous variables were presented as mean and standard deviation and the median with interquartile range as warranted. Shapiro-Wilk normality test was performed to test normality. Categorical variables were presented as numbers and percentages. The comparison for the evolution of AIG was performed using Chi-square test.

A Sankey diagram was included to illustrate the overall change of histological stage at the follow-up endoscopy. Moreover, a Kaplan Meier analysis was performed to evaluate the evolution into a worse histological stage.

Statistical analyses were performed using Stata (StataCorp. 2016. Stata Statistical Software: Release 13. College Station, TX: StataCorp LP). A $p<0.05$ was considered as statistically significant.

Results:

Participants

Out of 1004 patients with positive PCA, a total of 426 patients were included, 316 females with a median of 54.4 years (IQR 45.3-63.2). The detailed flow-chart is shown in figure 1. The most frequently associated symptoms were dyspepsia in 149 patients (35%) followed by vitamin B12 deficiency in 141 patients (33.º%). Autoimmune comorbidities were observed in 149 patients (35.5%). Among these, autoimmune hypothyroidism was the most prevalent, occurring in 18.7% of the cases. Previous helicobacter eradication was present in 80 patients (18.8%). The rest of the baseline characteristics can be observed in Table 1.

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The distribution of patients based on their PCA titers was as follows: 100 patients (23.7%) exhibited a titer of 1/160, 151 patients (35.5%) had a titer of 1/320, 111 patients (26.1%) presented with a titer of 1/640, and 64 patients (15%) displayed a titer of 1/1280.

Endoscopic and histological findings

Endoscopically 33 patients presented antral and corporal endoscopic signs of atrophy (8.1%), 57 patients presented corporal and fundic atrophy (13.9%), 36 patients (8.8%) antral, corporal and fundic atrophy while 69 patients presented exclusively corporal atrophy (16.9%).

Histologically, 105 patients were classified as potential AIG (24.7%), 99 patients as stage I (23,2%), 215 patients as stage II (50.5%) and 7 patients as stage III (1.6%). Therefore, 222 patients were considered as advanced AIG.

At the gastric body, 139 patients presented intestinal metaplasia (32.6%), 29 patients pseudopyloric metaplasia (6.8%) and 28 patients intestinal and pseudopyloric metaplasia (6.6%). Metaplasia was less frequent at the gastric antrum, as 49 patients presented intestinal metaplasia (11.5%), one patient pseudopyloric metaplasia (0.2%) and another patient intestinal and pseudopyloric metaplasia (0.2%).

The presence of intestinal/pseudopyloric metaplastic changes was more frequent in advanced stages (73.9% vs 26.1%; RR 2.8 (95% CI; 2.15-3.6, $p<0.001$). Low-grade dysplasia without visible lesions was detected in 3 patients.

Prevalence

During this period, a total of 26798 patients underwent at least one upper endoscopy, so the prevalence of AIG was estimated to be 1.6% (95% CI 1.4-1.7%). When only patients with advanced AIG were considered, the estimated prevalence was 0.8% (95% CI 0.7-0.9%) and 0.4% (95% CI 0.3-0.5%) for potential AIG.

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Evolution:

153 patients presented a subsequent endoscopy with biopsies according to Sydney's protocol and were considered for the follow-up. The baseline characteristics for these patients is shown in table 2. At the index gastroscopy, 75 patients (49%) showed no endoscopic evidence of atrophy, 8 patients (5.2%) with exclusive antral atrophy, 13 patients (8.5%) with antral and corporal atrophy , 22 patients with corporal and fundic atrophy (14.4%), 27 patients (17.7%) with exclusive corporal atrophy and 8 patients with atrophy of both antrum and gastric body and fundus (5.2%).

Forty-four patients (28.8%) were categorized as stage 0, 29 patients (19.5%) as stage I, 78 patients as stage II (51%) and 2 patients (1.3%) as stage III. The follow-up gastroscope was performed after a median of 35.1 months (IQR 25,5-47.2). 13 patients were classified as stage 0 (8.5%), 32 patients as stage I (20.9%), 103 patients as stage II (67.3%) and 5 patients as stage III (3.3%). Low-grade dysplasia without visible lesions were identified in 2 patients. Metaplastic changes were detected in 68 patients (44.4%) at the baseline endoscopy and in 82 patients at the follow-up endoscopy (53.6%) (diff 9.2% 95% CI -2.1%-20.4%: p=0.11) (table 3).

At the index gastroscopy 52.3% presented advanced GAI with a significant increase to 70.6% at the follow-up gastroscopy (difference 18.7% 95% CI 7.6-29%%; p=0.001). Also, a significant decrease of potential GAI into more advanced stages was observed at the follow-up gastroscope (26.8.% vs 8.5%; difference 20.2% 95% CI 11.8-28.7%; p<0.001) .).

Also, a detailed evolution at the follow-up endoscopy is shown in figure 2. Furthermore a Kaplan-Meier analysis into a worse histological stage is shown in figure 3.

Polyps, neuroendocrine tumours and adenocarcinoma

At baseline 39 patients exhibited hyperplastic polyps (9.2%), 8 patients fundic gland polyps (1.9%). 3 adenomas with low-grade dysplasia (0.7%) and 3 patients presented

G1 neuroendocrine tumours (0.7%). Additionally, one patient (0.2%) was diagnosed with signet-ring cell gastric carcinoma in advanced stage (T4N1M0).

At follow-up gastroscopy, 13 patients presented incident hyperplastic polyps (8.5%), two patients presented neuroendocrine tumours (1.3%) and 1 patient was diagnosed with antral adenocarcinoma (0.7%). The only patient diagnosed with adenocarcinoma was a T1bN0M0 and presented at the baseline a stage I

Discussion

Our study demonstrated a low prevalence of autoimmune gastritis (AIG) among patients undergoing upper gastrointestinal endoscopy. Notably, fifty percent of these patients presented with a relevant atrophy upon histopathological evaluation. Furthermore, we observed significant histological deterioration during the follow-up, even in patients with potential AIG. Despite these findings, the prevalence and incidence of gastric adenocarcinoma were very low.

The prevalence of 1.6% of all patients undergoing upper gastrointestinal endoscopy during a 10-year period is similar to those reported in other Western countries and Eastern countries. A retrospective study conducted at a single-center in the United States over the period from 1988 to 2008 reported an overall prevalence of AIG of 1.1%[8]. A multicenter European cross-sectional study, including 1123 individuals, identified a prevalence of 2.3%[9]. A Japanese study that included asymptomatic individuals, estimated a prevalence of 0.49% based on histological and serological markers[10]. These rate could increase, whenever only positivity to PCA are considered, as a Spanish study, that included a sample of 429 patients with a prevalence of 7.8%, particularly higher for postmenopausal women[11].

We found a low prevalence of gastric adenocarcinoma, with one case at baseline and another at the follow-up endoscopy. Massironi et al, performed a multicentric Italian cross-sectional study with AIG including a total of 612 patients and found that 2,1% presented adenocarcinoma [12]. These differences could be explained that this study included only patients with established diagnoses of atrophic stages. However, another recent large prospective study, with a median disease history of 18 years, did not report any incidence of gastric adenocarcinoma [7]. Thus, a second prospective study including 211 patients with AIG in naïve HP-negative patients, did not find any increased risk of gastric adenocarcinoma, hypothesizing that previous reported increased risk of gastric adenocarcinoma could be related to prior or current unrecognized HP infection [13]. Moreover, these rates are very similar to those reported for patients without the prevalence of gastroesophageal cancer is estimated to be less than 0,4% in subjects with endoscopic evaluation of dyspepsia [14].

At the follow-up, we observed an increase in the rate of advanced AIG and a decrease in the rate of potential AIG, clearly indicating and affirming that AIG is a progressive disorder. Furthermore, while autoimmune gastritis (AIG) has commonly been associated with the atrophy of the oxyntic mucosa, it is important to note that this condition also includes early stages of non-atrophic gastritis, as demonstrated in our study [15] [16]. The evolution of potential AIG into atrophic stages was first demonstrated by a prospective Italian study that included 51 patients with positive PCA and no histological changes, and 47.1% evolved into overt AIG in a median time of 2 years [4]. This statement reaffirms the current recommendation for endoscopic surveillance every 3 to 5 years, with a primary focus on the detection of neuroendocrine tumors rather than gastric adenocarcinoma[17] [18].

However, some limitations should be mentioned. Firstly, it is a retrospective evaluation and standardised or blind evaluation of biopsies by pathologists were not performed. Secondly, the lack of standardised follow-up endoscopies could overestimate the rate of patients with histological progression as there is a risk of selection bias of patients with worse symptoms to have their follow-up endoscopy earlier. Thirdly, other endoscopic signs of AIG as sticky adherent mucus, the type of remnant oxyntic mucosa or white global appearance were not recorded, as these findings could not be properly

assessed in the endoscopic records, although these findings might be only present in advanced stages [19] [20]. Fourthly, enterochromaffin cell proliferation is one of the histological signs of AIG but was not assessed in our study, although these findings might not have a relevant clinical impact. Fifth, we included only positive PCA AIG in our analysis, given that approximately 20% of patients with AIG are seronegative [21]. Unfortunately, we were unable to identify these patients.

In conclusion, AIG presented a low prevalence among patients undergoing gastroscopy. Biopsies according to Sydney's protocol in patients with positive parietal cell antibodies might adequately stage a progressive condition. Moreover, it is crucial to consider the full spectrum of both non-atrophic and atrophic stages, as up to fifty percent of individual might exhibit atrophy at baseline.

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Availability of data and materials: The data supporting the findings of this study are not openly available due to reasons of sensitivity and are available from the corresponding author upon reasonable request.

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AUTHOR CONTRIBUTIONS

Planning and/or conducting the study: EF-V,

Collecting data: SE-C, ACR, AGD, MdCL-M, IRdlP, JP-V, SB, ICM-F, BR-BA, RLM, LACH, AS, DAR.

Data analysis: EF-V

Drafting the manuscript: EF-V, JP-V, ABD.

All authors had access to the complete dataset and have reviewed and approved the final manuscript.

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Table 1. Baseline characteristics. GERD: gastroesophageal reflux disease; PSC: primary sclerosing cholangitis; IBD: inflammatory bowel disease.

	N= 426 patients.
Sex (female)	316 (74.4%)
Smoking habit	
No	290 (68.1%)
Former smoker	68 (16%)
Smoker	68 (16%)
History of gastric cancer	
First degree	12 (2.8%)
Second degree	7 (1.6%)
Previous HP eradication	80 (18.9%)
Associated symptoms	
Ferropenic anemia	43 (10.1%)

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Megaloblastic anemia	19 (4.5%)
Vitamin B12 deficiency	142 (33.3%)
Dyspepsia	149 (35%)
GERD	19 (4.5%)
Ferropenia	11 (2.6%)
Nausea/vomits	7 (1.6%)
Others	36 (8.5%)
Autoimmune comorbidity	
Rheumatoid arthritis	14 (3.3%)
PSC	8 (1.9%)
Type I Diabetes	13 (3.1%)
IBD	7 (1.6%)
Celiac disease	5 (1.2%)
Autoimmune hepatitis	2 (0.5%)
Sjögren syndrome	2 (0.5%)
Autoimmune thyroiditis	81 (19%)
Others	19 (4.5%)
Anti-parietal cell antibodies	
1/160	100 (23.5%)
1/320	151 (35.5%)
1/640	111 (26.1%)
1/1280	64 (15%)
Anti-intrinsic factor antibodies	2.7 (IQR 0.5-8.8)
Hemoglobin (g/dl)	13.6 (IQR 12.3-14.5)
Ferritin	34.5 (IQR 15-83)
Vitamin B12	284 (IQR 194.5-392.5)

Table 2. Baseline characteristics of all patients with follow-up endoscopy.

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	N= 153 patients.
Sex (female)	120 (78.9%)
Age, median in years (IQR)	51.6 (IQR 44.3-64.1)
Smoking habit	
No	121 (79.1%)
Former smoker	17 (11.1%)
Smoker	15 (9.8%)
History of gastric cancer	
First degree	3 (2%)
Previous HP erradication	27 (17.7%)
Anti-parietal cell antibodies	
1/160	30 (19.6%)
1/320	66 (43.1%)
1/640	33 (21.6%)
1/1280	24 (15.7%)
Anti-intrinsic factor antibodies	4 (IQR 0.5-13)
Hemoglobin (g/dl)	13.4 (IQR 12-14.2)
Ferritin	22 (IQR 11-51)
Vitamin B12	272 (IQR 190-381)

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Table 3. Evolution of autoimmune gastritis, comparing the index endoscopy and follow-up endoscopy. AIG: autoimmune gastritis

	Index endoscopy	**Follow-up endoscopy**	P value
Potential AIG	44 (26.8%)	13 (8.5%)	<0.001
Advanced AIG	80 (52.3%)	108 (70.6%)	0.001
Corporal metaplastic changes	68 (44.4%)	82 (53.6%)	0.11
Intestinal metaplasia	52 (34 %)	56 (36.6%)	
Pseudopyloric metaplasia	4 (2.6%)	10 (6.5%)	
Intestinal+pseudopyloric metaplasia	12 (7.8%)	16 (10.5%)	
Corporal dysplasia	1 (0.6%)	2 (1.2%)	0.9

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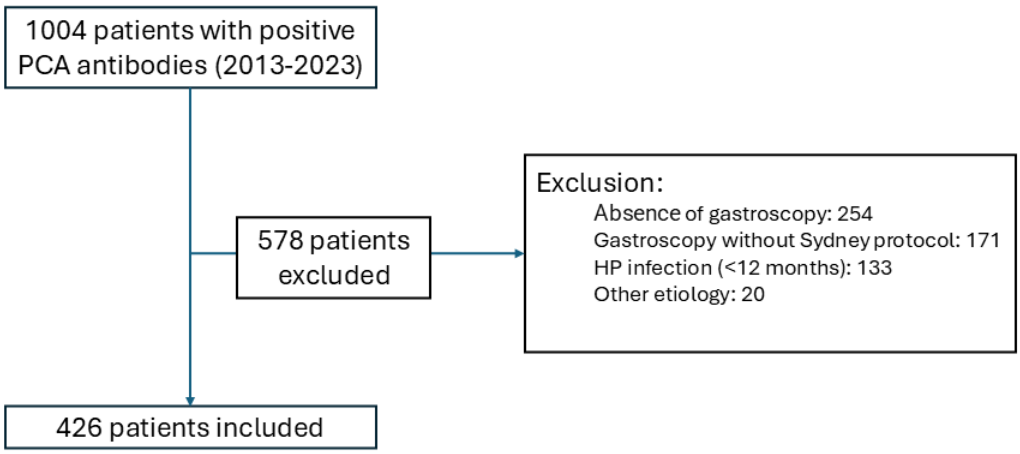

Figure 1. Flowchart of all included and excluded patients.

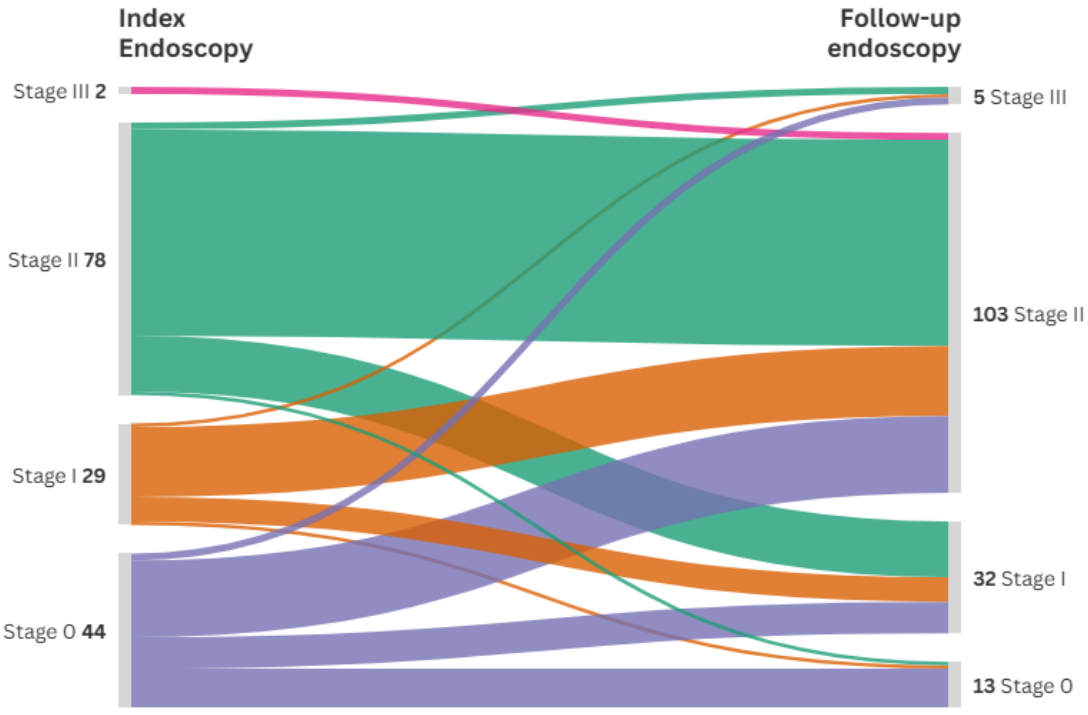

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Figure 2. Sankey diagram showing the evolution of the different stages at the follow-up endoscopy.

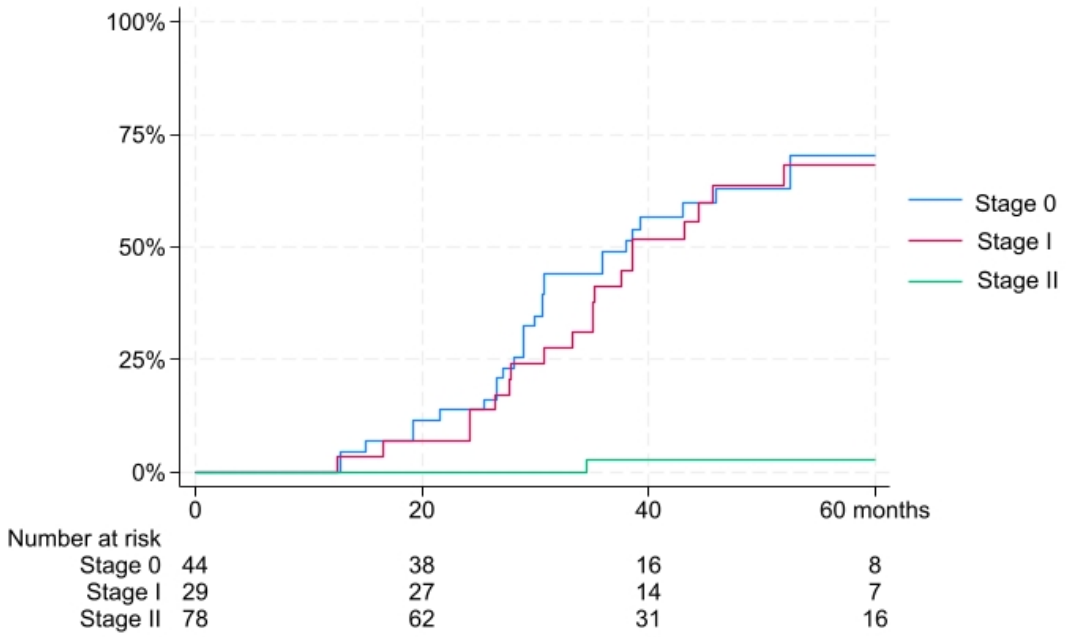

Figure 3. Kaplan-Meier analysis showing the evolution into a worse histological stage, stratified according to the baseline stage.