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RESEARCH ARTICLE

Helicobacter pylori and autoimmunity in atrophic gastritis - comparison of clinical, endoscopic and histopathological features

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Objective: This study aims to investigate the clinical, endoscopic, biologic and histopathological differences between Helicobacter pylori-associated and autoimmune gastric atrophy.

Methods: A retrospective analysis was conducted on 95 patients diagnosed with either H. pylori-related corporal and antral atrophy (43 patients) or autoimmune corporal atrophic gastritis (52 patients).

Results: A significant male predisposition for H. pylori-associated atrophic changes in both the antrum and corpus regions (p=0.007, OR=3.24) was observed in comparison with autoimmune etiology of atrophy. While comorbidities and lifestyle factors showed similar distributions across groups, only unintentional self-reported weight loss demonstrated a significant association with H. pylori atrophy (p=0.0177, OR=3.94). Corporal erosions were strongly associated with antral and corporeal atrophic gastritis (p=0.04, OR=8.27), but the rest of mucosal lesions are comparable among groups. Interestingly, patients with H. pylori-related pangastric atrophy exhibited lower frequencies of altered triglyceride (p=0.018) and cholesterol (p=0.029) levels compared to the autoimmune group. Linear regression analysis identified low triglyceride levels as an independent predictor for H. pylori-associated antral and corporal atrophic gastritis (p=0.04) in endoscopic population with atrophy, but no hematological or clinical parameters were predictive for these changes.

Conclusions: Male patients are more likely to present with corpus atrophic gastritis associated with H. pylori infection than with an autoimmune etiology. Patients with atrophic gastritis tend to have similar clinical characteristics, except for dyslipidemia, which is more prevalent in those with H. pylori pangastritis. Corporal erosions are associated with active H. pylori infection in atrophic mucosa.

Keywords: atrophic gastritis, H. pylori, autoimmune gastritis

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Introduction

Chronic atrophic gastritis, characterized by chronic inflammation and thinning of the stomach lining, has two commonly accepted etiologies, each one of them being responsible for a specific type of inflammation that can be recognized histologically [1]. These predominant forms are Helicobacter pylori-induced atrophic gastritis and autoimmune atrophic gastritis. Even though both of them have the common endpoint of gastric atrophy, their origins, progression, and potential complications differ substantially [2,3].

Helicobacter pylori (H. pylori) is an important source of chronic inflammation due to the interplay between the bacterium's virulence factors, the host's immune responses and various environmental influences that dictate a persistent inflammatory process that affects multiple regions of the stomach, including both the antrum and the oxyntic mucosa present in the corpus and fundus [4]. Over time, this chronic inflammation may cause the progressive destruction of parietal and chief cells, which are responsible for the production of gastric acid and digestive enzymes, respectively. The loss of these essential cell types results in the atrophy observed in H. pylori-induced atrophic gastritis [5,6].

In contrast, autoimmune-induced atrophic gastritis is driven by a misdirected immune response, where the body's immune system mistakenly attacks the healthy parietal and chief cells within the stomach lining, leading to selective destruction and atrophy, usually limited to the corpus and fundus regions of the stomach, leading to loss of gastric acid production [3,7,8]. This condition typically develops gradually over an extended period, often without noticeable symptoms in its early stages. As it progresses, patients may experience abdominal pain, nausea, vomiting, and severe bloating. Serious consequences of autoimmune atrophic gastritis are related to nutrient deficiencies, particularly vitamin B12 and iron, which can result in pernicious anemia and other serious systemic complications [9]. These nutrient deficiencies arise due to the complete destruction of parietal cells, which are responsible for the production of intrinsic factor, a crucial compound necessary for the absorption of vitamin B12. Additionally, the

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complete loss of acid-secreting parietal cells can significantly impair the absorption of iron, leading to debilitating iron deficiency anemia [10]. Patients with autoimmune atrophic gastritis may also develop other autoimmune disorders, further complicating their clinical presentation and management.

Autoimmune gastritis, or type A gastritis, primarily affects the corpus and fundus of the stomach. The histological hallmarks of this condition include a dense lymphocytic infiltrate in the lamina propria, often extending to the base of the gastric glands, and prominent lymphoid aggregates. As the disease advances, there is a selective loss of parietal and chief cells in the oxyntic mucosa, leading to atrophy. This is frequently accompanied by the emergence of intestinal metaplasia and pseudopyloric metaplasia [11,12].

Conversely, in H. pylori-induced gastritis, also known as type B gastritis, the initial impact is primarily on the antrum of the stomach. This condition is marked by a superficial inflammatory infiltrate rich in plasma cells, accompanied by lymphoid follicles. Over time, these changes can lead to atrophy of the antral mucosa and the development of intestinal metaplasia. The involvement of the corpus typically occurs as a later event in the disease progression [13,14].

This article provides a comprehensive exploration of demographic, clinical, biologic and endoscopic findings in patients with H. pylori-induced corporal and antral atrophic gastritis (CAG + AAG) in comparison to those presenting autoimmune corporal atrophy (CAG).

Methods

Patient's selection

The single-center retrospective study enrolled 95 consecutive patients admitted in the Medical Clinic No. 2 of the Emergency County Hospital located in Targu Mures, Romania, between January 2016 and June 2024. All patients underwent gastroscopic evaluation due to dyspepsia (manifested as heartburn, epigastric pain, postprandial fullness, abdominal meteorism, nausea, vomiting), appetite decrease, unintentional weight loss, or anemia. In order to evaluate the severity and frequency of these symptoms, a modified version of the Leeds Dyspepsia Questionnaire was used [15]. Strict exclusion criteria were implemented. Therefore, 1942 patients who were underage or diagnosed with any form of neoplasia, end-stage hepatic or renal failure, dementia, or inflammatory bowel disease were excluded, as well as those whose data regarding chronic comorbidities, medication, social behavior, endoscopic, or histopathologic results were incomplete.

Our study included 43 patients with H. pylori-related antral and corporal atrophic gastritis (AAG + CAG – *H. pylori)* and 52 patients with corporal atrophic gastritis (CAG – Autoimmune) determined by autoimmune etiology (confirmed by histopathologic results and serologic tests). (Figure 1)

Data collection

The clinical presentation was carefully documented, with particular attention paid to key symptoms associated with

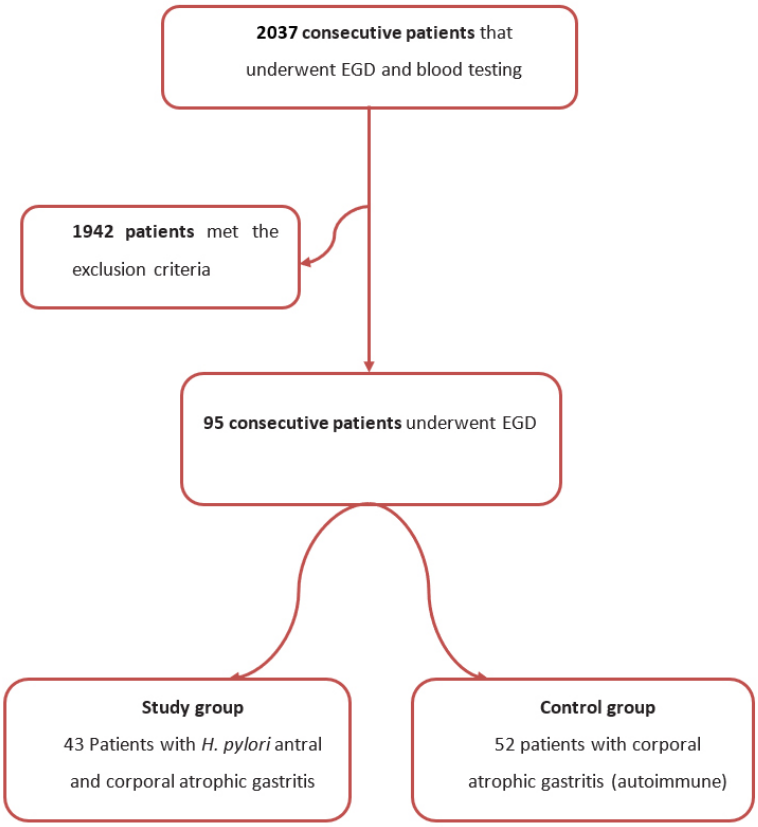

Fig. 1. The selection of patients

gastrointestinal distress, comorbidities, and chronic medication.

Laboratory results included hemoglobin levels with gender-specific normal ranges established as 12–17 g/dL for women and 13–17 g/dL for men; mean corpuscular volume (MCV): with a normal range of 78–95 fl for both genders; and serum iron levels: with gender-specific normal ranges of 9.0–30.4 mmol/L for women and 11–28 mmol/L for men; International Normalized Ratio (INR): 0.8–1.1; fibrinogen: 2–4.0 g/dL;. total cholsterol levels: <5.17 mmol/L; triglyceride levels: < 1.7 mmol/L;

Medication history was carefully documented, with a focus on two key categories: proton pump inhibitors (PPIs), such as pantoprazole, esomeprazole, and omeprazole use prior to endoscopy, and non-steroidal anti-inflammatory drugs (NSAIDs). The consumption of potentially gastrotoxic drugs, primarily diclofenac and ibuprofen, was noted based on patient medical records, as well as anticoagulant and antiplatelet therapy. Lifestyle factors were also taken into account, with particular attention paid to smoking habits and alcohol consumption.

The study was approved by the Ethics Committee of the Emergency County Hospital of Targu Mures, Romania nr. 3119 /29.04.2024.

Data regarding esophagogastroduodenoscopy (EGD) and histopathological analysis

Upper digestive endoscopy was performed on each patient, and we closely observed and documented visible mucosal lesions (erosions, defined as mucosal defects measuring less than 5 mm in diameter; submucosal hemorrhages, indicating bleeding beneath the mucosal layer; and ulcers, characterized as defects exceeding 5 mm in size and extending into deeper tissue layers). The updated Sydney system was used for gastritis staging [16,17].

Five gastric biopsies were obtained and examined from each patient in accordance with the protocol outlined in the updated Sydney System (two antral, two corporal, and one angular sample). All biopsy specimens were dispatched in separate containers (based on the anatomical origin of the tissue sample), and they underwent standardized processing protocols, starting with fixation in a 10% buffered formalin solution, dehydration, paraffin embedding, and tissue sectioning using a microtome (3-5 μm). The following histological staining was used: Hematoxylin and eosin for general morphological analysis, periodic acid-Schiff-Alcian blue for the identification of intestinal metaplasia, and Giemsa stain for the detection of H. pylori microorganisms [18].

The histopathological analysis of these biopsies revealed that 43 subjects (45,26%) presented multifocal atrophic changes confined to both the antral and corporeal mucosa, along with the presence of H. pylori, while 52 patients (54,74%) exhibited atrophic gastric mucosa limited exclusively to the corpus, with the antrum remaining unaffected and positive serological testing for anti-parietal-cell antibodies.

Statistical analysis

We realized the statistical analysis using Graphpad Prism 8.0.2 Software. The distribution of the continuous variables was non-parametric (as indicated by the Shapiro-Wilk test for normality), therefore median value and interquartile range were used to express quantitative data. Categorical variables were analyzed using absolute values and percentages. The Mann-Whitney test was applied in order to analyze the difference between these groups. The significance limit was set as $\alpha = 0.05$. Odds ratios (OR) were determined in order to determine the associations between the variables. Linear regression models were used to analyze predictors for *H. pylori* inflicted CAG and AAG.

The ones that were statistically significant ($p<0.05$) were included in the multiple regression analysis in order to determine the independent predictors of H. pylori related antral and corporal atrophy.

Results

Gender-based analysis of the groups revealed a higher predisposition for male patients to develop atrophic changes in both the antrum and corpus regions of the stomach ($p=0.007$, OR=3.24. Older patients (>65 years old) did not present statistically significant differences in these groups. (Table 1)

Comorbidities, including cardiovascular, respiratory, chronic kidney diseases, and diabetes mellitus, exhibited similar distributions across both groups ($p>0.05$ for all conditions). Cardiovascular diseases were highly prevalent, affecting both groups equally (86.05% and 86.54%). (Table 1)

Lifestyle factors such as smoking and alcohol consumption (Table 1), as well as chronic medication use (Table 2), did not show statistically significant associations with atrophic changes in the gastric mucosa ($p > 0.05$ for all comparisons).

The prevalence of gastrointestinal symptoms (epigastric pain, heartburn, nausea/vomiting, diarrhea) showed no statistically significant differences between the two groups ($p>0.05$ for all comparisons). However, weight loss demonstrated a significant association with pangastric atrophy ($p=0.017$, OR = 3.94). (Table 3)

Endoscopic examination revealed no significant associations between gastric atrophy and the presence of erythematous gastritis localized in the antrum ($p=0.78$) or corpus ($p>0.99$), antral gastric ulcers ($p>0.99$), submucosal hemorrhages ($p=0.79$), or hiatal hernia ($p>0.99$). However, antral and corporeal atrophic gastritis demonstrated strong associations with corporal erosions of the mucosa ($p=0.04$, OR=8.27). Intestinal metaplasia was more prevalent in patients with *H. pylori*-related antral and corporal atrophic gastritis (97,67% and 90,38%, respectievly) without reaching statistical significance ($p=0.21$). (Table 4)

A statistically significant association was observed be-

Table 1. Data regarding demographical and clinical features in patients with AAG+CAG (H. pylori) and CAG (autoimmune)

Parameter	Total	AAG + CAG (H. pylori)		CAG (autoimmune)		p-value	OR	95% CI
		n	%	n	%			
Age > 65 years	72	33	76,74%	39	75,00%	>0,99	1,1	0,43 - 2,77
Male	47	28	65,12%	19	36,54%	0,007	3,24	1,33 - 7,26
Female	48	15	34,88%	33	63,46%	-	-	-
Comorbidities								
Cardiovascular diseases	82	37	86,05%	45	86,54%	>0,99	0,95	0,31 - 3,29
Cerebrovascular diseases	8	4	9,30%	4	7,69%	>0,99	1,23	0,33 - 4,44
Chronic respiratory diseases	25	13	30,23%	12	23,08%	0,48	1,44	0,59 - 3,57
Diabetes mellitus	14	5	11,63%	9	17,31%	0,56	0,62	0,21 - 2,09
Arterial hypertension	69	34	79,07%	35	67,31%	0,25	1,83	0,69 - 4,40
Liver diseases	14	5	11,63%	9	17,31%	0,56	0,62	0,21 - 2,09
Osteoarticular disorders	16	5	11,63%	11	21,15%	0,27	0,49	0,17 - 1,45
Chronic kidney disease	13	5	11,63%	8	15,38%	0,76	0,72	0,24 - 2,18
Anemia	41	16	39,02%	25	60,98%	0,30	0,64	0,28 - 1,42
Hashimoto's thyroiditis	6	1	2,33%	5	9,62%	0,21	0,22	0,01- 1,78
Lifestyle factors								
Smoking	15	6	13,95%	9	17,31%	0,78	0,77	0,24 - 2,40
Alcohol	12	7	16,28%	5	9,62%	0,36	1,82	0,56 - 5,59

Abbreviations: AAG, antral atrophic gastritis; CAG, corporal atrophic gastritis; CI, confidence interval; OR, odds ratio;

Table 2. Data regarding chronic medication use in patients with AAG+CAG (H. pylori) and CAG (autoimmune)

Parameter	Total	AAG + CAG (H. pylori)		CAG (Autoimmune)		p-value	OR	95% CI
		n	%	n	%			
Antiplatelet drugs	31	13	30,23%	18	34,62%	0,66	0,81	0,36 - 1,94
Non-antivitamin K oral anticoagulants	19	11	25,58%	8	15,38%	0,30	1,89	0,68 - 5,41
Antivitamin K oral anticoagulants	6	1	2,33%	5	9,62%	0,21	0,22	0,01 - 1,78
ACEI	43	22	51,16%	21	40,38%	0,30	1,54	0,69 - 3,41
Betablockers	46	25	58,14%	21	40,38%	0,10	2,05	0,87 to 4,64
NSAIDs	29	14	32,57%	15	28,85%	0,82	1,19	0,49 - 2,97
PPIs	52	21	48,84%	31	59,62%	0,30	0,64	0,29 to 1,43

Abbreviations: AAG, antral atrophic gastritis; ACEI, Angiotensin-converting enzyme inhibitors; CAG, corporal atrophic gastritis; CI, confidence interval; NSAIDs, Nonsteroidal anti-inflammatory drugs; OR, odds ratio, PPIs, proton pump inhibitors

Table 3. Data regarding clinical manifestations in patients with AAG+CAG (H. pylori) and CAG (autoimmune)

Parameter	Total	AAG + CAG (H. pylori)		CAG (autoimmune)		p-value	OR	95% CI
		n	%	n	%			
Epigastric pain/ discomfort	33	13	30,23%	20	38,46%	0,51	0,69	0,30 - 1,61
Heartburn	9	2	4,65%	7	13,46%	0,17	0,31	0,063 - 1,58
Nausea/ Vomiting	11	3	6,98%	8	15,38%	0,33	0,41	0,11 - 1,52
Loss of appetite	13	8	18,60%	5	9,62%	0,24	2,14	0,70 - 6,34
Diarrhea	4	3	6,98%	1	1,92%	0,32	3,82	0,54 - 50,51
Weight loss	18	13	29,55%	5	9,62%	0.01	3,94	1,22 - 10,67

Abbreviations: AAG, antral atrophic gastritis; CAG, corporal atrophic gastritis; CI, confidence interval; OR, odds ratio

Table 4. Group differences regarding endoscopic and histologic features in patients with AAG+CAG (H. pylori) and CAG (Autoimmune)

Parameter	Total	AAG + CAG (H. pylori)		CAG (Autoimmune)		p-value	OR	95% CI
		n	%	n	%			
Endoscopic findings								
Gastric resection	4	3	6,98%	1	1,92%	0,32	3,82	0,54 - 50,51
Hiatal hernia	16	7	16,28%	9	17,31%	>0,99	0,92	0,34 - 2,73
Erythematous mucosa								
Antral	79	35	81,40%	44	84,60%	0,78	0,79	0,27-2,33
Corporal	25	11	25,58%	14	26,92%	>0,99	0,93	0,36 - 2.23
Erosive gastritis								
Antral	29	14	32,56%	15	28,30%	0,66	1,22	0,48 - 3,04
Corporal	7	6	13,95%	1	1,92%	0,04	8,27	1,22 - 96,51
Gastric ulcers								
Antral	7	3	6,98%	4	7,69%	>0,99	0,9	0,21 - 3,51
Submucosal hemorrhages	18	9	20,93%	9	17,31%	0,79	1,265	0,46 - 3,45
Duodenal lesions	8	5	11,63%	3	5,77%	0,46	2,149	0,51 - 8,45
Diffuse duodenal white deposits	11	4	9,30%	7	13,46%	0,74	0,65	0,20 - 2,34
Histopathologic findings								
Intestinal metaplasia	89	42	97,67%	47	90,38%	0,21	4,46	0,55 - 53,74

Abbreviations: AAG, antral atrophic gastritis; CAG, corporal atrophic gastritis; CI, confidence interval; OR, odds ratio

tween pangastric atrophy and alterations in serum lipid profiles. Specifically, patients with atrophy in both corpus and antrum exhibited a lower frequency of changes in triglyceride (p=0.01) and cholesterol (p=0.02) levels compared to autoimmune group. Hematological parameters, including hemoglobin, mean corpuscular volume (MCV), and serum iron levels, showed no statistically significant differences between the groups (p>0.05 for all comparisons). However, descriptive statistics revealed slightly lower mean hemoglobin levels and MCV ((85.2 fL vs 88.5 fl) in the *H. pylori* group (11.2 g/dL) compared to the autoimmune group (12.2 g/dL). (Table 5)

Linear regression models questioned the presence of independent predictors for *H. pylori* induced-antral and corporal atrophic gastritis, but proved that only low levels of triglycerides remained an independent predictor for the H pylori associated AAG + CAG (p=0.04). (Table 6)

Discussion

The high predisposition of male patients to develop atrophic changes in both the antrum and corpus (p=0.007, OR=3.24) is well known and it could be attributed to several factors, such as hormonal influences, lifestyle factors, and the immune response to H. pylori infection. Estrogen has been shown to have protective effects on the gastric mucosa [19]. The lower levels of estrogen in males might contribute to their increased susceptibility to atrophic changes. Regarding lifestyle factors, men may be more likely to engage in behaviors that increase the risk of gastric mucosal damage, such as higher rates of smoking or alcohol consumption [20]. Additionally, some studies suggest that men may have higher rates of H. pylori infection or may be more susceptible to its damaging effects [21].

Related to clinical presentation, our findings indicate that there are no specific symptoms that can reliably predict atrophic pangastritis. This observation aligns with previous research that reported that the clinical presentation of atrophic gastritis can be highly variable and often asymptomatic[22]. Similarly, Jones and Lee (2018) found that many patients with histologically confirmed atrophic pangastritis did not exhibit any characteristic symptoms [23].The lack of predictive symptoms poses a significant challenge for early diagnosis and intervention due to delayed detection of atrophic pangastritis, potentially increasing the risk of progression to more severe conditions such as gastric cancer.

However, a significant association between weight loss and pangastric atrophy (p=0.01, OR = 3.94) highlights the potential systemic effects of extensive atrophic changes. This relationship could be explained by several mechanisms (malabsorption, altered gastric motility, increased production of inflammatory mediators). Atrophic changes may lead to decreased production of intrinsic factor, impacting vitamin B12 absorption [24], and it affects gastric emptying, potentially leading to early satiety and reduced food intake [25]. Iron, folate, and other micronutrient deficiencies associated with atrophic gastritis could contribute to weight loss [26]. Chronic inflammation associated with atrophic gastritis may lead to increased production of cytokines that can affect appetite and metabolism [27].

Our analysis revealed no statistically significant associations between the etiology of atrophic gastritis and the various comorbidities, lifestyle factors, and chronic medication use examined in this study, as opposed to previous data. The lack of significant associations across these categories suggests that the etiology and risk factors for atroph-

Table 5. Comparison of median values and interquartile range (Q1-Q3) of laboratory results in patients with AAG+CAG (H. pylori) and CAG (autoimmune)

Parameter	AAG + CAG (H. pylori) Median value (Q1-Q3)	CAG (autoimmune) Median value (Q1-Q3)	p-value
Hemoglobin	11,2 (8,80-13,5)	12,20 (10,38-13,68)	0,17
MCV	85,20 (78,60-92,70)	88,55 (84,93-94,73)	0,06
Serum iron levels	11,5 (6,00-15,90)	12,15 (6,85-17,90)	0,45
Triglycerides	1,08 (0,69-1,45)	1,28 (0,94-1,65)	0,018
Fibrinogen	3,91 (3,01-4,85)	3,7 (3,19-4,64)	0,63
Cholesterol	3,68 (2,81-4,57)	4,38 (3,28-5,06)	0,02
Serum glucose	5,91 (5,01-6,95)	5,85 (5,24-6,55)	0,91
INR	1,26 (1,04-1,485)	1,12 (1,0-3,32)	0,09

Abbreviations: AAG, antral atrophic gastritis; CAG, corporal atrophic gastritis; CI, confidence interval; INR, international normalized ratio; MCV, mean corpuscular volume; OR, odds ratio

Table 6. Results from multivariate regression model analyzing the predictive value of laboratory parameters in AAG+CAG (H. pylori) and CAG (autoimmune)

Model 1	B	SE	Beta	t	p
Hemoglobin	-0.003	0.021	-0.018	-0.144	0.886
MCV	-0.008	0.006	-0.205	-1.466	0.148
Serum Iron	0.011	0.009	0.178	1.178	0.243
Total Cholesterol	-0.079	0.042	-0.244	-1.893	0.063
Triglycerides	-0.228	0.110	-0.277	-2.065	0.043
Glucose	0.023	0.034	0.082	0.687	0.494
Fibrinogen	0.037	0.038	0.117	0.964	0.338
INR	0.064	0.062	0.118	1.044	0.300

Abbreviations: AAG, antral atrophic gastritis; CAG, corporal atrophic gastritis; CI, confidence interval; INR, international normalized ratio; MCV, mean corpuscular volume; OR, odds ratio

ic gastritis may include confounding factors not accounted for in our study. Notably, our study population exhibited a high prevalence of cardiovascular diseases, probably favored by chronic inflammation [28].

Only one patient from each group had corporal ulcers; therefore, data analysis is irrelevant. The strong associations between atrophic gastritis and corporal erosions (p=0.04, OR=8.27) confirm that atrophic changes are a consequence of persistent inflammation [29,30].

The higher prevalence of antral reactive gastropathy in patients with predominant corporal atrophy (p<0.0001, OR=0.01) supports the concept of topographical progression in atrophic gastritis proposed by Correa (1988) and suggests a potential compensatory mechanism [31]. Reduced acid secretion from the atrophic corpus may lead to hypergastrinemia, stimulating the antral mucosa and resulting in reactive changes. This pattern may represent an intermediate stage in the progression towards pangastritis [32].

The high prevalence of intestinal metaplasia across study groups, while not statistically significant, emphasizes the importance of implementing risk stratification strategies based on the extent and severity of atrophic changes and intestinal metaplasia and exploring potential strategies to reduce the risk of progression to gastric cancer in high-risk individuals [33,34].

A meta-analysis by Pormohammad et al. (2018) found that H. pylori infection was associated with a 2.24-fold increased risk of gastric cancer [35]. Although less prevalent than H. pylori-induced atrophy, corpus-restricted atrophy has been associated with a 3- to 6-fold increased risk of gastric cancer compared to the general population [36]. This elevated risk is particularly noteworthy given the specific location of the atrophy. Furthermore, the loss of parietal cells in the corpus leads to hypergastrinemia, which in turn increases the risk of developing gastric neuroendocrine tumors, especially type 1 gastric NETs [37].

The differences in cancer types between H. pylori-induced and autoimmune atrophy can be attributed to the distinct pathophysiological mechanisms involved. H. pylori infection induces chronic inflammation and oxidative stress throughout the stomach, leading to widespread mucosal damage and genetic alterations [38]. In contrast, autoimmune atrophic gastritis primarily affects the corpus and fundus, with relative sparing of the antrum, resulting in a different pattern of mucosal changes and subsequent neoplasia [39].

H. pylori is a known trigger of autoimmune diseases, including autoimmune thyroiditis, gastritis or rheumatoid arthritis [40]. Even though H. pylori gastritis and autoimmune gastritis are different entities, gastric autoimmunity can develop in some subjects during bacterial infection (due to genetic predisposition) [41].

These findings highlight the need for tailored surveillance strategies based on the etiology of gastric atrophy. While both groups require regular monitoring, patients with H. pylori-induced atrophy may benefit from more frequent endoscopic surveillance and potentially more aggressive management of precancerous lesions. Anti-H. pylori treatment administration reduces significantly the incidence of gastric cancer, as a study that enrolled 180,284 patients confirms, especially if the eradication therapy is successful [42].

The analysis of inflammatory markers in our study revealed trends that, although not reaching statistical significance, align with the established relationship between pangastric atrophy and inflammatory status. Some studies reported alterations in various blood markers associated with atrophic gastritis, particularly in the context of H. pylori infection [43]. Similarly, patients with autoimmune gastritis often exhibit changes in hematological parameters, reflecting the chronic inflammatory state characteristic of this condition. It is important to note that while our findings suggest a potential link between pangastric atrophy and altered hematological parameters, caution should be exercised in interpreting these results. As emphasized by Neumann et al. (2018), the association between gastric pathology and systemic inflammatory markers is multifaceted and can be influenced by various confounding factors [44].

The association between corporal atrophy and alterations in serum lipid profiles (triglycerides: p=0.01; cholesterol: p=0.02) is a novel finding that opens up new avenues for research. The lower frequency of changes in triglyceride and cholesterol levels in patients with more extensive atrophy (both corpus and antrum) may be explained by the complex interplay between gastric function and lipid metabolism. The stomach plays a crucial role in the digestion and absorption of nutrients, including lipids. Gastric atrophy can lead to decreased acid production and altered gastric motility, potentially affecting lipid absorption and metabolism [45]. One possible explanation for our findings is that more extensive gastric atrophy may lead to a more pronounced impairment of gastric function, resulting in decreased lipid absorption and, consequently, less variation in serum lipid levels. This hypothesis is supported by studies demonstrating that gastric atrophy is associated with malabsorption of various nutrients, including fats [46].

The observed trends in hematological parameters, while not statistically significant, are consistent with the known association between atrophic gastritis and iron-deficiency anemia [32]. The lack of statistical significance could be due to variations in the duration or severity of atrophic gastritis among study participants or potential iron supplementation in some patients, masking the true impact of atrophic changes on iron status.

The small sample size and the focus on a population from a single center (with similar social and demographic characteristics) can limit the validity of our study across different populations. Serum gastrin and chromogranin A levels, as well as H. pylori antibodies, were not studied due

to their unavailability in selected patients. Despite these limitations, our findings are valuable due to lack of data in the literature regarding clinical, endoscopic, and histopathological findings associated with chronic gastric atrophy. Further research is needed in order to understand its implications.

Conclusion

Male patients referred for endoscopy are more likely to present with corpus atrophic gastritis associated with H. pylori infection than with autoimmune etiology. Patients with atrophic gastritis tend to have similar clinical and biological characteristics, except for unintentional self-reported weight loss and dyslipidemia, which are more prevalent in those with H. pylori pangastritis compared to those with autoimmune gastritis. There are no significant differences in hematological parameters of patients with different mechanisms of corpus atrophy, but corpus mucosal erosions are associated with H. pylori induced atrophy.

Authors' contribution

AMF (Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Validation; Visualization; Writing – original draft; Writing – review & editing)

SNM (Conceptualization; Formal analysis; Investigation; Project administration; Supervision; Writing – original draft; Writing – review & editing)

SM (Data curation; Investigation; Project administration; Resources; Validation)

DH (Data curation; Investigation; Methodology; Writing – original draft;)

MP (Data Investigation; Project administration; Resources; Validation)

AN (Conceptualization; Formal analysis; Investigation; Project administration; Resources; Supervision; Validation; Visualization; Writing – original draft; Writing – review & editing)

Conflict of interest

None to declare.

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