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Autoimmune gastritis: clinical and histological study in a Peruvian population

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Abstract

Summary The diagnosis of autoimmune gastritis poses significant challenges, particularly in resource-limited settings where access to serological tests is restricted. This study aimed to evaluate the histological, endoscopic, and serological features of patients diagnosed with autoimmune gastritis in our population.

Methods We retrospectively reviewed cases diagnosed with autoimmune gastritis at two medical centers in Peru. Clinical data, serological and endoscopic reports were collected for each case, and gastric mucosal tissue samples from the antrum and corpus were histologically examined. Immunohistochemistry was also performed to evaluate neuroendocrine hyperplasia.

Results Histologically, all 44 cases exhibited atrophy in the corpus, with the majority presenting at advanced stages of the disease (84%). However, endoscopic findings did not correlate with histology, as only 59.09% of cases showed corpus atrophy on endoscopy. Immunohistochemical analysis revealed neuroendocrine hyperplasia in all cases (100%). Anti-intrinsic factor antibody was positive in only 25% of cases, whereas 84.1% showed positivity for anti-parietal cell antibodies.

Conclusion Histological evaluation of autoimmune gastritis cases demonstrates significant diagnostic potential, offering an effective alternative to costly and less accessible serological tests, particularly in resource-limited settings like ours.

Keywords Autoimmune gastritis, Histological findings, Endoscopic findings, Serological studies, Anti-intrinsic factor antibody, Anti-parietal cell antibody

Introduction

Autoimmune gastritis (AIG) is a complex immunological disease with a wide range of reported prevalence, varying from 0.3 to 19.5% [1–4]. This discrepancy can be attributed to the challenges in diagnosing the disease [5]. Moreover, AIG is considered the most common cause of atrophic gastritis worldwide [6]. In Latin America, AIG is a rarely diagnosed disease, with few published studies focusing primarily on clinical and endoscopic findings [7–10].

A study from Brazil reported a 1.4% prevalence of "corpus-restricted atrophic gastritis, likely autoimmune,"

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based on histological findings without additional serological studies [7]. In contrast, a Chilean study found that 7.5% of cases with atrophic gastritis were AIG, although serological studies were only performed in four cases [8].

AIG is characterized by the destruction of parietal cells, mediated by circulating antibodies against H+/K + ATPase and intrinsic factor, as well as sensitized T lymphocytes [5, 11, 12]. This destruction leads to hypochlorhydria and achlorhydria, resulting in intrinsic factor loss and subsequent vitamin B12 deficiency [13–15]. The hypochlorhydric environment also promotes G cell hyperplasia in the antral mucosa, increasing serum gastrin levels and potentially leading to the proliferation of endocrine-type cells in the corpus, which may give rise to neuroendocrine tumors [13, 16, 17].

The diagnosis of AIG is based on serological, clinical, and pathological findings. Gastroscopy with separately collected antrum and corpus biopsies, along with classic histological findings, is the gold standard [3, 12]. Serological findings include the study of anti-parietal cell and anti-intrinsic factor antibodies, gastrin-17, and the ratio or index between Pepsinogen I and II [5, 18]. However, antibody tests are not used as a gold standard due to variability in sensitivity and specificity, and seronegative AIG cases may be encountered [4].

The histological findings of autoimmune gastritis (AIG) vary gradually depending on the stage of the disease, which can be early, florid, or advanced [5, 19–22]. In general, AIG is characterized by severe involvement of the gastric mucosa of the corpus, compared to the antral mucosa, which typically shows minimal inflammation and variable G-cell hyperplasia [14]. The typical histological picture of AIG includes glandular atrophy and intestinal metaplasia in the corpus mucosa, pseudopyloric metaplasia and pancreatic acinar metaplasia, basal mononuclear inflammation, and enterochromaffin-like (ECL) cell neuroendocrine hyperplasia [14–16, 23–27]. In the end stage, AIG is characterized by severe atrophic oxyntic mucosa with marked epithelial metaplasia and ECL-cell hyperplasia, without prominent inflammation [19].

In recent years, we have observed an increasing number of gastric corpus biopsies exhibiting histological features of AIG. These patients typically present with anemia associated with dyspepsia and lack prior serological studies. This study aims to determine the histological, endoscopic, and serological features of patients with AIG in a Peruvian population, thereby increasing awareness of this pathology in our country.

Methods

This was a retrospective, cross-sectional descriptive study which was conducted at the Hospital Nacional Daniel Alcides Carrion (HNDAC) and a Private Pathology Center, both located in Lima-Peru; from 2019 to 2024. From which medical records from patients with the diagnosis of AIG were selected.

During the 2023–2024 period, the Private Center for Digestive Pathology processed an annual average of 9,097 gastric biopsies. Among these, gastritis associated with *Helicobacter pylori* accounted for 62.9% of cases, subdivided into non-atrophic gastritis (46.0%) and atrophic gastritis (16.9%). In contrast, autoimmune gastritis had a prevalence of 0.30%, corresponding to 28 cases per year.

At the Daniel Alcides Carrión Hospital, during the same period, an annual average of 1,279 gastric biopsies was processed. Gastritis related to *H. pylori* was identified in 53.5% of cases, while autoimmune gastritis reached a prevalence of 0.75%, equivalent to 10 cases per year.

In the present study, a case of AIG was defined as the presence of the following histological changes: greater atrophic involvement of the corpus, in relation to the antrum mucosa; *Helicobacter pylori* negative, and oxyntic mucosa with at least one of the following features: Intestinal metaplasia, pseudopyloric metaplasia and neuroendocrine hyperplasia.

The inclusion criteria for our study were the following:

1. Stomach biopsies that include both the antrum and corpus.
2. Endoscopy reports available.
3. Histological features described in the case definition.
4. Serological study report of at least one of the following: anti-parietal cell antibodies or anti-intrinsic factor antibodies, regardless of whether the result was positive or negative. This criterion ensured that antibody testing was performed in all included patients, even though three cases had negative results for both markers.
5. Immunohistochemistry stain positivity with at least one of the following: Chromogranin and Synaptophysin.
6. Sufficient histological material to perform new sections and immunohistochemistry stains.

We excluded cases with only biopsies of the corpus and with insufficient clinical data.

The medical ethical committees of HNDAC, and the Private Pathology Center approved the study protocol. Informed consent was not required as the study was not covered under WMO (Medical Research Involving Human Subjects Act).

The following clinical data was collected in all the patients recruited: age, sex, serological study for AIG, associated diseases, hematological findings, and endoscopic findings classified as the following: fundic atrophy,

antrum atrophy, pan atrophy, no atrophy and others (Such as polyps or mucosal erosion).

The stomach tissue (biopsy) from these patients was subjected to histological examination and then immunohistochemistry (Chromogranin or Synaptophysin) was performed to evaluate neuroendocrine hyperplasia.

The histologic features were assessed in hematoxylin and eosin (H&E) slides. The microscopic features included were: (1) Glandular atrophy defined as a loss of glandular tissue [28]. (2) Intestinal metaplasia morphologically recognized by the presence of goblet cells, absorptive cells and cells resembling colonocytes [28]. (3) Pseudopyloric metaplasia: defined as a pyloric gland mucosa located where oxyntic mucosa should be [28–30]. (4) Pancreatic acinar cell metaplasia: morphologically composed of pancreatic acinar cells in stomach characterized by cytoplasm that shows acidophilic and granular in the apical and middle compartment and basophilic in the basal, arranged in lobules [28, 30]. (5) Polymorphonuclear neutrophil activity: defined as the presence of neutrophils with a variable density in the gastric mucosa [28]. (6) Eosinophils operationalized as the number of eosinophils per high power field in the gastric lamina propria, in normal conditions the presence of these cells in the gastric mucosa is minimal (1–3 for HPF) [31, 32]. (7) Lymphoid aggregated or lymphoid follicles, the first one defined as a group of lymphocytes and plasma cells without germinal center and the lymphoid follicles as an aggregated of lymphocytes with germinal center [33]. All the histological findings found in antrum were also described.

The immunohistochemical study to confirm neuroendocrine hyperplasia was performed with Bio SB polyclonal antibody for Synaptophysin and Bio SB monoclonal antibody for Chromogranin. Cells with positive cytoplasmic staining were considered as positive for neuroendocrine cell. Neuroendocrine hyperplasia was classified as linear or micronodular according to the following criteria. Micronodular hyperplasia: A group of 5 or more neuroendocrine cells bounded by basement membrane with a diameter of less than 150 μm. Linear hyperplasia: at least 2 columns of 5 or more neuroendocrine cells [5, 34].

Duodenal biopsies were assessed according to standard criteria: lymphocytic duodenitis was defined as ≥ 25 intraepithelial lymphocytes per 100 enterocytes with preserved villous architecture, and atrophic duodenitis with intraepithelial lymphocytosis as villous atrophy associated with increased intraepithelial lymphocytes. Celiac disease was not systematically ruled out by serology, since this test is not routinely performed in our hospital prior to biopsy, and in some cases the information was not available in the clinical record. Thus, celiac disease or autoimmune enteropathy could not be excluded or confirmed, as these diagnoses were not the objective of our study [35].

Descriptive statistics were used to report our findings.

Table 1 Clinical data of the 44 AIG cases included in the study

Clinical characteristics of the study group	
Age in years (mean ± standard deviation)	59.3 ± 17.18
Male: Female	1:2
Associated diseases:	
Hypothyroidism	4
Diabetes Mellitus 2	2
Hematological findings	
Vitamin B12 decreased	30 (68.18%)
Anemia	22 (50%)
Serological studies	
Total number of positive cases for Anti-parietal cell antibody	37 (84.1%)
Total number of positive cases for Anti-intrinsic factor antibody	11 (25%)
Positivity for both antibodies at the same time	7 (15.9%)

Results

A total of 94 cases with the diagnosis of AIG were evaluated, out of which 44 cases fullfilled the inclusion and exclusion criteria and were recruited in the study. We found a greater prevalence of AIG in women compared to men, in a 2:1 ratio. The average age was approximately 59.3 years, and the oldest and youngest were 86 and 33 years respectively. 30 (68.18%) of the patients tested positive just for Anti-parietal cell antibody, 4 (9.09%) tested positive only for Anti-intrinsic factor antibody, 7 (15.9%) for both serological markers at the same time, and only 3 (6.82%) cases were negative for both markers. (Table 1).

Microscopically, all cases exhibited body atrophy, with severe atrophy observed in the majority of cases (84%). (Fig. 1) Neuroendocrine hyperplasia, confirmed by immunohistochemistry for Chromogranin or Synaptophysin, was also present in all cases, manifesting as both micronodular and linear in most cases (97.7%) (Fig. 2). And the great majority had also pseudopyloric metaplasia (97.7%). (Fig. 3). Histological findings in antral mucosa were identified. (Table 2). Also, duodenal biopsy was assessed in 15 cases of AG.(Table 3).

Discussion

This study aimed to identify the histological, endoscopic, and serological features of autoimmune gastritis (AIG) in a Peruvian population. Most cases (84%) exhibited advanced histological stages of AIG, with atrophy present in all cases. However, endoscopic reports only detected atrophy in 59.09% of cases. Serological tests revealed that 84.1% of cases were positive for anti-parietal cell antibodies, while only 25% were positive for anti-intrinsic factor antibodies.

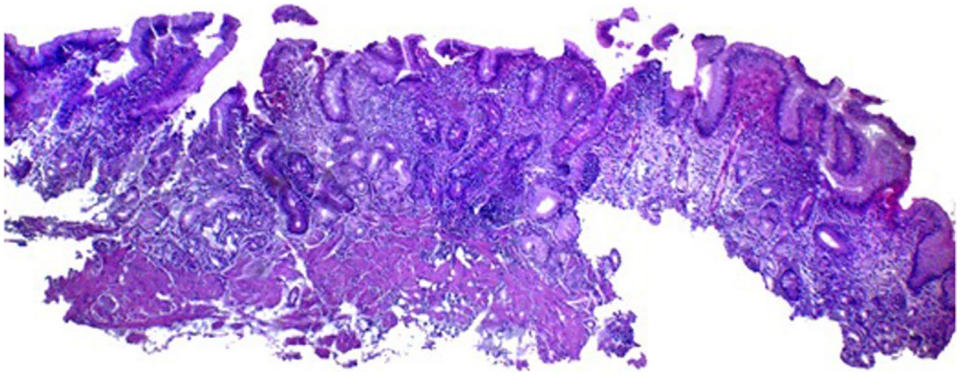

Fig. 1 Panoramic photo of the atrophic corpus mucosa with intestinal and pseudopyloric metaplasia (HE 4X)

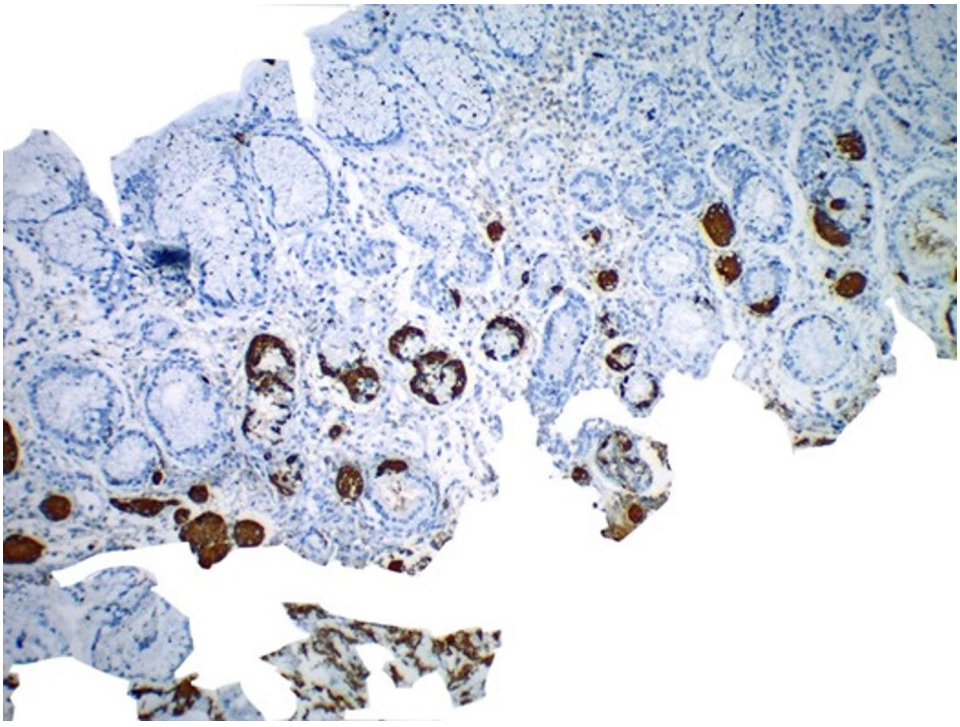

Fig. 2 Corpus mucosa with nodular neuroendocrine hyperplasia, and next to it, pseudopyloric metaplasia (HE 40X)Micronodular and linear neuroendocrine hyperplasia marked with immunohistochemical staining (Chromogranin 10X)

Endoscopic findings in AIG

Endoscopic reverse atrophy is a characteristic finding in autoimmune gastritis (AIG), present in approximately 85% of patients [14, 36–41]. However, in our study, only 34.09% of cases showed this pattern of reverse atrophy, and furthermore, 41% of cases did not detect atrophy in the corpus at all. This is a disturbing finding, as the diagnostic suspicion of AIG should arise during endoscopy. It is unclear why the endoscopic reports showed a low percentage of reverse atrophy in our study. However, it is likely that the lack of staff skills, a poor condition of endoscopic equipment, or a problem with typing the report may be involved.

The endoscopic findings also showed frequent total (antrum-corpus) gastric atrophy (25%), and 13.6% reported atrophy only in the antrum. (Table 4; Fig. 4) Although these data are consistent with other studies [38, 42], they cannot explain the low rate of reverse atrophy or the poor histopathological correlation observed in our study. Polyps are another endoscopic finding in AIG, present in 20% of cases [37–39]. In our study, the presence of polyps was rare (11.3%). Additionally, dense and sticky mucus was not found in any of the cases studied, probably since this finding is relatively new and unknown [37, 38, 43].

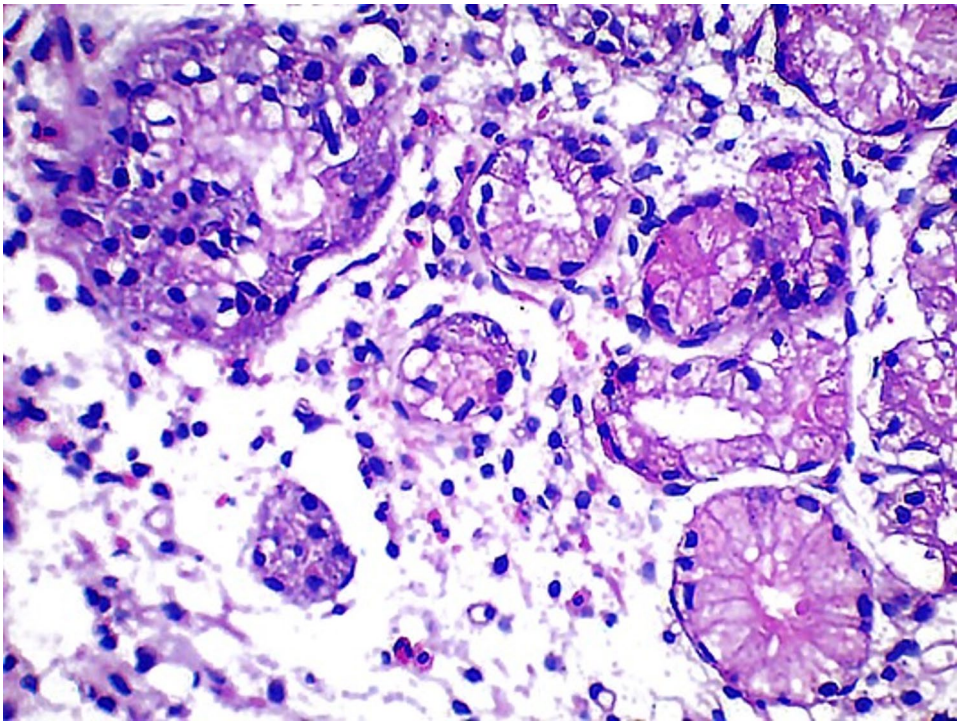

Fig. 3 Corpus mucosa with nodular neuroendocrine hyperplasia, and next to it, pseudopyloric metaplasia. (HE 40X)

Table 2 Autoimmune gastritis. Histologic findings in the antral mucosa

Histologic findings	Number (percentage)
Atrophy with intestinal metaplasia	4 (9.09%)
Preserved antral mucosa	11 (25%)
Reactive gastropathy	19 (43.18%)
Non atrophic gastritis. Helicobacter pylori negative	10 (22.73%)

Table 3 Autoimmune gastritis. Histologic findings in the duodenal mucosa

Histologic findings	Number (percentage)
Nonspecific duodenitis	5 (11.36%)
Atrophic duodenitis with intraepithelial lymphocytosis	4 (9.09%)
Lymphocytic duodenitis	6 (13.64%)
Absence of duodenal tissue for histological evaluation	29 (65.91%)

Table 4 Autoimmune gastritis. Endoscopic findings

Endoscopic pattern	Number (percentage)
Pan atrophy	11(25%)
Corpus atrophy only	15 (34.09%)
Antrum atrophy only	6 (13.64%)
No atrophy	12 (27.27%)

Histological features of AIG

The most notable histological features of advanced autoimmune gastritis (AIG) include inflammation, severe elongation of gastric pits, severe corpus atrophy with marked fibrosis, widespread epithelial metaplasia, and

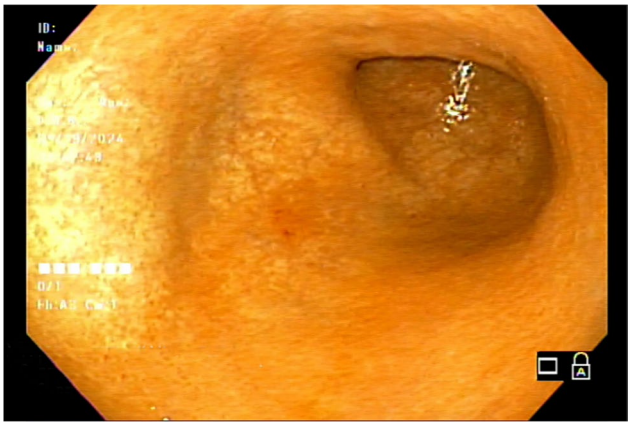

Fig. 4 Gastric body mucosa showing visible submucosal vessels and absence of folds

neuroendocrine hyperplasia [5, 19, 20, 22, 39, 41]. Our study found that all cases exhibited histological changes consistent with advanced AIG, including moderate to severe atrophy with marked metaplasia, minimal inflammation, and presence of neuroendocrine hyperplasia. However, it is essential to note that there is currently no standardized AIG grading system, which can lead to overlapping grades between stages [39].

Our study found a higher rate of pseudopyloric and intestinal metaplasia (97.73% and 90.91%, respectively) compared to other studies [27, 44–46]. This difference may be attributed to the advanced stage of our cases. Additionally, we found that complete intestinal

metaplasia was more prevalent than incomplete metaplasia (86.36% vs. 4.55%) [16, 46]. The incidence of pancreatic acinar metaplasia in our study (4.55%) was lower than expected [27, 42]. (Table 5)

Eosinophils were elevated (in some cases up to 40 HPF), but the average was 9 HPF, which is within the normal range [32]. This is lower than the average reported by other authors. Some studies suggest that eosinophilic infiltration is more prominent in the initial stages of AIG [22], which may explain the low eosinophil presence in our advanced stage cases. (Table 5).

Neuroendocrine hyperplasia was observed in almost all cases, with both linear and micronodular patterns. This type of hyperplasia is produced by gastrin stimulation on the enterochromaffin cells of the gastric corpus mucosa [5, 19, 22, 27, 39, 47]. Immunohistochemical studies with chromogranin and synaptophysin were positive in

Table 5 Autoimmune gastritis. Histologic findings in the corporal mucosa

Histologic findings	Number (percentage)
Atrophy	44 (100%)
Intestinal metaplasia	40 (90.9%)
Pseudopyloric metaplasia	43 (97.73%)
Pancreatic acinar metaplasia	2 (4.55%)
Neutrophil activity	21 (47.73%)
Eosinophils (mean ± standard deviation)	9.29 (23.2)
Lymphoid follicle	28 (63.64%)
Neuroendocrine hyperplasia	
Only nodular	0
Only linear	1 (2.27%)
Both nodular and linear	43 (97.73%)

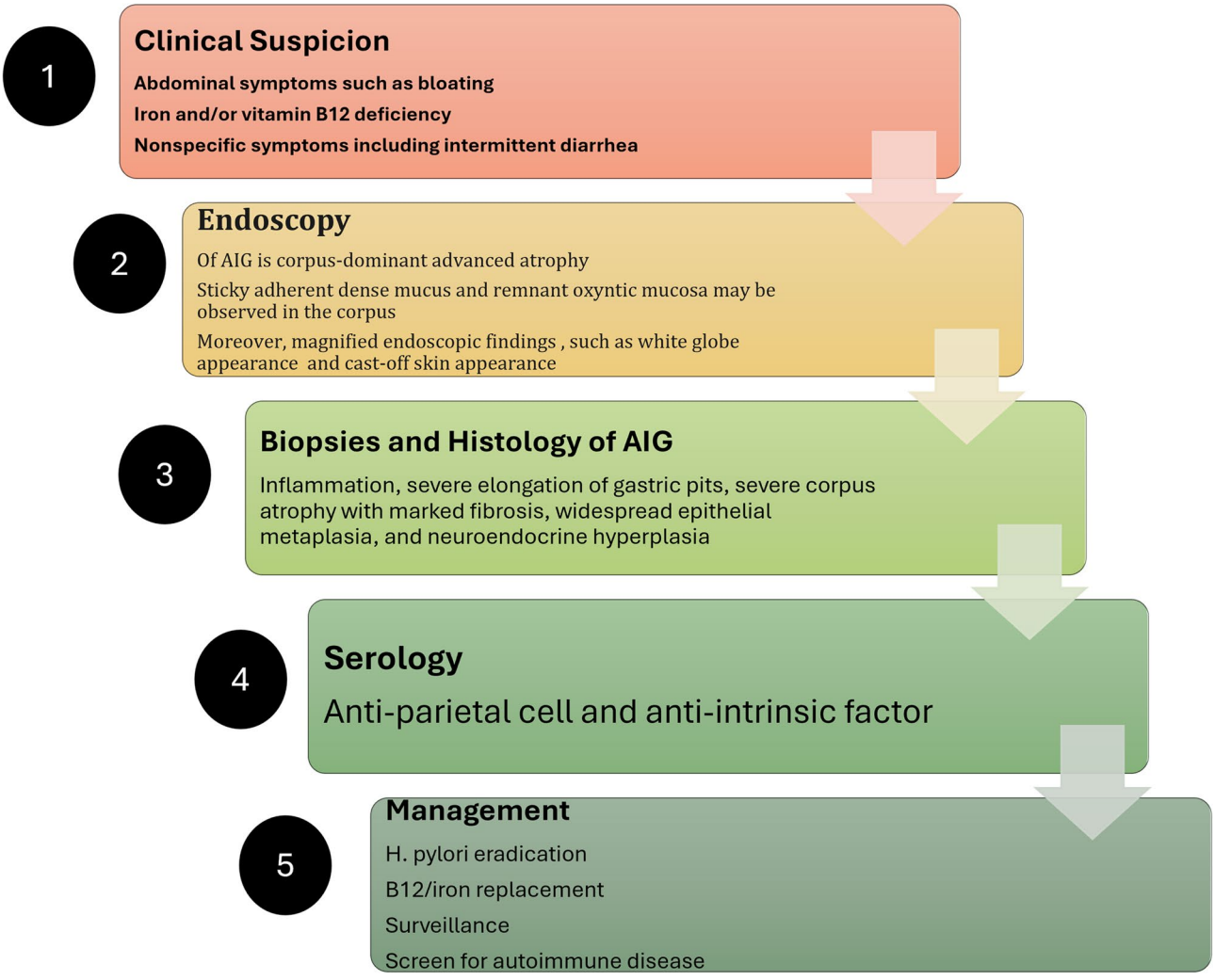

Fig. 5 Flow diagram starting off with clinical suspicion/symptoms, endoscopy findings, biopsy/histology findings, adjunct/serology markers and treatment

neuroendocrine cells, with chromogranin being more intensely positive than synaptophysin.

Changes corresponding to reactive gastropathy were identified in the gastric antrum mucosa of 19 out of 44 patients, which is consistent with findings from other studies [27]. (Table 2).

Serological findings in AIG

The positivity rates for anti-parietal cell and anti-intrinsic factor antibodies were 84.1% and 25%, respectively. These results are consistent with previous studies [23, 38, 48–50]. However, we expected a higher positivity rate in serological tests, given the advanced stage of most cases [51–53]. Interestingly, 3 (6.82%) of cases had negative results.

Two factors may contribute to the discrepancy in serological results. Firstly, the advanced stage of the disease may play a role, as anti-parietal cell antibody concentrations may decrease with progressive mucosal destruction [39, 54, 55]. Secondly, seronegative subjects are often elderly, with a higher prevalence in patients over 50 years [56]. This highlights the importance of considering population characteristics when studying AIG.

Anemia in AIG

Anemia in AIG may result from impaired iron absorption or intrinsic factor deficiency, leading to vitamin B12 deficiency [4]. Our study found an average hemoglobin level of 11.8 mg% and an anemia prevalence of 50%, consistent with previous reports [4, 57, 58]. However, vitamin B12 deficiency was more frequent in our cases (68.18%) than in other studies [59], possibly due to the advanced histological stage of our cases.

Our study had some limitations such as the small sample size, the absence of an integrated computerized record of the health system in our country, and the retrospective nature of the study, which precludes systematic prospective follow-up, as such, definitive conclusions cannot be drawn. Nevertheless, the findings provide a representative overview of the state of AIG in our region.

In conclusion, despite these limitations, our study showed the great diagnostic potential of histology in cases of AIG, especially in regions such ours where the costs of serological studies are high and moreover do not show positivity in all the cases studied. This discordance between histology and endoscopy highlights the importance of systematic gastric sampling, particularly in patients with anemia, since subtle histologic findings of autoimmune gastritis may be overlooked by endoscopic evaluation alone.

Regarding management of these patients, we follow the international guidelines [Shah et al., Gastroenterology 2021]. Patients receive a monthly dose of Vitamin B12, as well as folic acid or iron according to individual needs. Helicobacter pylori is searched for in all patients and eradication therapy is initiated as soon as the diagnosis is established. Patients are scheduled for regular endoscopic and histological follow-ups, especially in those with advanced atrophy, intestinal metaplasia, or type 1 gastric neuroendocrine tumors [60]. To facilitate clinical application, these recommendations have been summarized in the proposed diagnostic and management algorithm (Fig. 5).

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12876-025-04363-3.

Supplementary Material 1

Authors' contributions

FA: Study design •JF, EM, JM, PM: Collecting the cases and performing endoscopies. •FA, SR, RR, RR, GR: Interpretation of hystoloc samples. •All authors are responsible for the discussion, conclusion and approval of the final draft.

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Data availability

All data generated or analysed during this study are included in this published article and in the system [and its supplementary information files].

Declarations

Ethics approval and consent to participate

The present study was approved by the Ethics Committee of Hospital Nacional Daniel A. Carrión and Histodiagnostico Gastrointestinal. Private Pathology Center.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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