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Case Report

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Non-necrotizing gastric granulomas in a patient with biopsy-proven celiac disease and persistent hilar lymphadenopathy: A case report

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Abstract

Background: Celiac disease typically presents with diarrhea and malabsorption, though atypical presentations are increasingly recognized. Gastric abnormalities, particularly lymphocytic gastritis, have been described in association with celiac disease. However, non-necrotizing granulomatous inflammation is not a recognized gastric manifestation of celiac disease.

Case presentation: We report a 53-year-old man who presented with a one-year history of altered bowel habits characterized by decreased frequency (from ~2 bowel movements/day to ~1 every 2 days), recurrent epigastric pain, and fatigue, notably without diarrhea or weight loss. Endoscopy revealed scalloping of the duodenal mucosa with a grossly normal stomach. Duodenal biopsies demonstrated Marsh 3C celiac disease with total villous atrophy, crypt hyperplasia, and increased intraepithelial lymphocytes (>30/100 enterocytes). Gastric biopsies, obtained despite normal endoscopic appearance, unexpectedly revealed well-formed non-necrotizing granulomatous inflammation. Investigations, including colonoscopy and abdominal MRI, showed no evidence of Crohn's disease or other intra-abdominal pathology. Review of previous chest CT imaging examination revealed persistent hilar lymphadenopathy on images from 2022, 2025, and May 2026, raising the possibility of underlying sarcoidosis with gastric involvement . The patient's gastrointestinal symptoms improved following initiation of a gluten-free diet.

Conclusion: This case highlights the diagnostic challenge of non-necrotizing gastric granulomas in a patient with an atypical presentation of biopsy-proven celiac disease and persistent hilar lymphadenopathy. The combination raises suspicion for gastric involvement by sarcoidosis, although definitive diagnosis was not established. The coexistence of celiac disease and suspected sarcoidosis also raises the possibility of an underlying association between these immune-mediated disorders.

Keywords: Celiac disease, granulomatous gastritis, Marsh 3C, atypical presentation, hilar lymphadenopathy, gastric sarcoidosis, case report.

Introduction

Celiac disease is an immune-mediated enteropathy triggered by gluten ingestion in genetically susceptible individuals, affecting approximately 1% of the global population [1]. While diarrhea and malabsorption remain classic presenting features, the clinical spectrum has expanded considerably, with many patients presenting with non-classical symptoms including abdominal pain, fatigue, anemia, osteoporosis, and neurologic manifestations [2,3].

Gastric involvement in celiac disease has been increasingly recognized, with lymphocytic gastritis representing the most commonly described histologic abnormality. Studies have demonstrated a significant association between celiac disease and lymphocytic gastritis, particularly in patients with more severe villous atrophy [4]. Other gastric findings include

chronic active and chronic inactive gastritis, though these are generally considered part of the broader inflammatory spectrum rather than specific to celiac disease [5].

Non-necrotizing granulomatous gastritis, in contrast, is a rare histologic finding, reported in approximately 0.01–0.35% of gastric biopsies [6]. The differential diagnosis is broad and includes Crohn disease, sarcoidosis, infections (particularly mycobacterial and fungal), foreign-body reactions, autoimmune systemic diseases, and idiopathic granulomatous gastritis [7]. Granulomatous inflammation in the gastrointestinal tract has been described in association with celiac disease, though primarily in the ileocolonic region, raising the possibility of a mechanistic relationship between these 2 conditions [8]. Still, gastric granulomatous inflammation in the setting of biopsy-proven celiac disease appears to be exceptionally uncommon, with limited precedent in the literature. Because sarcoidosis is an important cause of non-necrotizing granulomatous inflammation and may involve the stomach, the identification of persistent hilar lymphadenopathy in a patient with gastric granulomas raises the an additional possibility of systemic sarcoidosis with gastrointestinal involvement.

We present a case of a 53-year-old man with biopsy-proven Marsh 3C celiac disease and concomitant non-necrotizing granulomatous gastritis, presenting with atypical bowel symptoms. We discuss the diagnostic challenges posed by this unusual combination and the possibility that the gastric granulomatous inflammation represents gastrointestinal involvement of suspected sarcoidosis.

Case Presentation

A 53-year-old man presented with an approximately one-year history of persistent alteration in bowel habits associated with recurrent epigastric abdominal pain. Before symptom onset, his usual bowel frequency was approximately two bowel movements per day. During the preceding year, the frequency decreased to approximately one bowel movement every 2 days, which was considered a significant change by the patient from his usual pattern. Importantly, he reported no episodes of diarrhea during this period. He denied hematemesis, melena, hematochezia, persistent nausea, vomiting, dysphagia, or odynophagia. There was no history of unintentional weight loss or significant loss of appetite. However, he reported fatigue that affected his daily activities.

The patient's past medical history was unremarkable, with no known chronic medical illnesses. In particular, there was no known history of hypothyroidism, diabetes mellitus, inflammatory bowel disease, or previously diagnosed celiac disease. There was no significant previous surgical history. The patient reported no known family history of gastrointestinal disease. He had no known history of chronic systemic or autoimmune diseases. The remainder of the available medical history was noncontributory.

On examination, the patient was conscious and clinically stable with normal vital signs. He was not cachectic and showed no clinical evidence of pallor, jaundice, or peripheral lymphadenopathy. Examination of the hands revealed no leukonychia, koilonychia, palmar

erythema, or clubbing. Abdominal examination revealed localized tenderness in the epigastric region, with no other significant abdominal abnormalities, organomegaly, or masses identified.

Laboratory investigations were unremarkable. Complete blood count demonstrated a hemoglobin level of 14.8 g/dL, with no evidence of anemia. Serum electrolytes were within normal limits. Liver enzymes were within the reported reference ranges. HbA1c was 5.2% and C-reactive protein was 1.2 mg/L, both within normal limits. Thyroid-stimulating hormone was within normal limits, with no biochemical evidence of hypothyroidism. Celiac serology demonstrated a strongly positive anti-tissue transglutaminase IgA screen of >200 U/mL (Table 1).

Table 1. Laboratory investigations at presentation

Parameter	Result	Reference Range
Hemoglobin	14.8 g/dL	13.0–17.0 g/dL
Hematocrit	45.2%	38.0–50.0%
MCV	88.1 fL	80.0–100.0 fL
WBC	6.9 K/µL	4.0–11.0 K/µL
Platelets	185 K/µL	150–400 K/µL
Sodium	137 mmol/L	135–145 mmol/L
Potassium	4.1 mmol/L	3.5–5.0 mmol/L
Chloride	100 mmol/L	98–106 mmol/L
AST	35 U/L	<40 U/L
ALT	33.8 U/L	<40 U/L
HbA1c	5.2%	<5.7%
CRP	1.2 mg/L	<5.0 mg/L
TSH	0.7029 mIU/mL	0.35–4.94 mIU/mL
Anti-tTG IgA	>200 U/mL	<15 U/mL

MCV: mean corpuscular volume; WBC: white blood cell count; AST: aspartate aminotransferase; ALT: alanine aminotransferase; CRP: C-reactive protein; TSH: thyroid-stimulating hormone; anti-tTG: anti-tissue transglutaminase.

Esophagogastroduodenoscopy (EGD) was performed, with normal appearing esophagus. The stomach also looked grossly normal without visible mucosal abnormalities, though biopsies were obtained to rule out gastritis. The duodenum demonstrated scalloping of the mucosa, and biopsies were obtained from the first and second parts of the duodenum to evaluate for celiac disease.

Colonoscopy revealed large internal hemorrhoids in the anus and rectum. The sigmoid colon, descending colon, and ascending colon appeared normal. In the transverse colon, a diminutive polyp was removed by cold snare and discarded. The cecum and terminal ileum appeared normal. No biopsies were obtained during colonoscopy, and no evidence of Crohn disease (including aphthous ulcers, strictures, or fistulas) was identified.

Microscopic examination of the duodenal specimens demonstrated features of celiac disease, Marsh classification 3C (Figure 1). Findings included total villous atrophy with crypt hyperplasia. There were increased intraepithelial lymphocytes, with more than 30 lymphocytes per 100 enterocytes. The lamina propria showed mild active inflammation with focal eosinophil infiltrates of up to 20 eosinophils per high-power field. No evidence of microorganisms, granulomas, dysplasia, or malignancy was identified. The absence of granulomas in the duodenal biopsy, despite their presence in the stomach, is noteworthy.

On microscopic examination of the gastric mucosa, well-formed non-necrotizing granulomatous inflammation was noted (Figure 2). The differential diagnosis provided by the pathologist included autoimmune systemic disease, sarcoidosis, foreign-body reaction, and infectious etiology. Special stains, including Ziehl-Neelsen for acid-fast bacilli and periodic acid-Schiff for fungal organisms, were recommended to further evaluate infectious causes. H. pylori was not identified on routine staining.

Given the unexpected finding of gastric granulomas, further investigations were pursued. Colonoscopy demonstrated no endoscopic evidence of Crohn disease, with a normal terminal ileum and no ileocolonic pathology. A subsequent abdominal MRI showed no significant abdominal abnormality, including no evidence of intra-abdominal lymphadenopathy, masses, or bowel wall thickening. Review of the patient's presvious chest CT subsequently revealed persistent hilar lymphadenopathy on studies performed in 2022, 2025, and May 2026 (Figure 3). These finding in light of well-formed non-necrotizing granulomas, raised the possibility of sarcoidosis with gastric involvement. Further diagnostic evaluation for sarcoidosis was discussed, however, the patient did not pursue additional investigations.

Following the diagnosis of celiac disease, the patient was initiated on a strict gluten-free diet. At follow-up, he reported significant improvement in his gastrointestinal symptoms, including resolution of epigastric pain and normalization of bowel frequency. However, repeat gastric biopsy was not performed, and therefore the response of granulomatous inflammation to gluten withdrawal could not be assessed. No specific treatment was initiated for the granulomatous gastritis because no definitive underlying diagnosis has been established and given the patient did not pursue further evaluation.

Discussion

Celiac disease is classically characterized by diarrhea, weight loss, and features of malabsorption. However, the clinical presentation has evolved substantially, with many patients presenting with non-classical or even asymptomatic disease [2]. Our patient presented with constipation-predominant altered bowel habits without diarrhea, which is distinctly atypical for severe Marsh 3C celiac disease. This pattern highlights the importance of maintaining a low threshold for investigating celiac disease even in patients without classic diarrheal symptoms, particularly when other features such as fatigue or abdominal pain are present.

The absence of anemia, nutritional deficiencies, or weight loss in our patient despite severe histologic changes is also noteworthy. This underscores the variable penetrance of celiac disease manifestations and the dissociation between histologic severity and clinical phenotype. The strongly positive anti-tTG IgA serology (>200 U/mL) confirmed the immunologic basis of the enteropathy, consistent with the Marsh 3C changes observed on duodenal biopsy.

Gastric involvement in celiac disease has been well documented in the literature. Lymphocytic gastritis is the most commonly recognized gastric manifestation, with studies demonstrating a strong association between celiac disease and this histologic finding, particularly in patients with more severe villous atrophy [4]. The pathogenesis of gastric involvement in celiac disease remains incompletely understood. Some authors have proposed that the gastric inflammatory changes may represent a local manifestation of the same immunologic process that affects the duodenum, possibly in response to gluten exposure [9,10]. This hypothesis is supported by reports of histologic improvement in gastric inflammation following gluten-free diet [5]. However, granulomatous inflammation is not a recognized gastric manifestation of celiac disease. The presence of well-formed non-necrotizing granulomas in the gastric mucosa of our patient represents an unusual finding that extends beyond the typical spectrum of celiac-associated gastritis.

Granulomatous gastritis is a rare histologic finding, with reported prevalence ranging from 0.01% to 0.35% of gastric biopsies [6]. The differential diagnosis is broad and requires careful clinical correlation. Crohn disease is one of the most common causes, with granulomas identified in up to 30% of isolated gastric biopsies from patients with Crohn disease [11]. Sarcoidosis is another important consideration, with gastric involvement reported in 10% of patients with systemic sarcoidosis, though symptomatic gastric sarcoidosis occurs in 0.1-0.9% of the cases [12].

Infectious etiologies must also be excluded, particularly mycobacterial (tuberculosis, atypical mycobacteria) and fungal infections (histoplasmosis, candidiasis) [13]. Foreign-body reactions to food particles, suture material, or medications can also produce granulomatous inflammation. Autoimmune systemic diseases such as granulomatosis with polyangiitis and systemic lupus erythematosus are additional considerations [14].

Idiopathic granulomatous gastritis is a diagnosis of exclusion, accounting for a substantial proportion of cases after thorough investigation [7]. Our patient's evaluation did not identify an alternative etiology, though complete exclusion of all causes could not be established.

In our patient, the presence of persistent hilar lymphadenopathy raises the possibility of sarcoidosis. When considered alongside with well-formed non-necrotizing granulomas identified in the gastric mucosa, these findings raise the possibility that the gastric granulomas represent gastrointestinal involvement of systemic sarcoidosis. Sarcoidosis is a multisystem granulomatous disorder of unknown etiology that commonly involves the lungs and mediastinal lymph nodes [15]. Gastrointestinal involvement is relatively uncommon, with symptomatic gastric sarcoidosis being particularly rare [12]. However, granulomas can be found incidentally in gastric biopsies of patients with systemic sarcoidosis, and the stomach can appear endoscopically normal despite histologic involvement. The diagnosis of gastric sarcoidosis requires clinical and radiological

correlation, exclusion of other granulomatous diseases, and, when possible, tissue confirmation from an involved site.

An additional consideration is whether the coexistence of celiac disease and suspected sarcoidosis in this patient represents more than coincidental disease. Several case reports and small series have described an association between celiac disease and sarcoidosis, raising the possibility of shared immune dysregulation [16,17]. Both diseases involve T-cell-mediated inflammatory responses and have been linked to similar HLA haplotypes [17]. However, the reported association remains controversial, and population-based studies have yielded conflicting results [18]. We cannot establish causality from a single observation, but the coexistence in our patient is consistent with previously reported cases.

Granulomatous gastrointestinal inflammation has previously been reported in association with celiac disease. Johns et al. described granulomatous ileocolitis in a patient with celiac disease and raised the possibility of an association between the two conditions [8]. Their report is relevant to the present case because it provides precedent for granulomatous gastrointestinal inflammation occurring in the setting of celiac disease, although the distribution differs substantially. In contrast, the granulomas in our patient were identified in the stomach, while the presence of persistent hilar lymphadenopathy raises the alternative possibility of systemic sarcoidosis.

The central question raised by this case is the etiology of the gastric granulomas. We propose several possible explanations:

First, the gastric granulomas may represent gastrointestinal involvement of sarcoidosis. Given the persistent hilar lymphadenopathy demonstrated on three separate chest CT examinations over four years, this is now the most clinically compelling hypothesis. The non-necrotizing nature of the granulomas, the lack of infectious microorganisms identified on routine histologic examination, and the lack of endoscopic evidence of Crohn disease are all consistent with this diagnosis.

Second, the granulomas could represent an independent granulomatous gastritis coinciding with celiac disease, including infectious, foreign-body, or idiopathic granulomatous gastritis. The prevalence of celiac disease (approximately 1%) makes coincidental occurrence statistically plausible, though granulomatous gastritis itself is rare.

Third, the granulomas could represent an extremely rare manifestation of celiac disease, analogous to the ileocolonic granulomas reported by Johns et al. [8]. This would suggest that the spectrum of celiac disease may extend beyond the duodenum to include granulomatous inflammation in other parts of the gastrointestinal tract. However, this hypothesis is challenging to prove without repeat biopsy following gluten withdrawal, and the presence of persistent hilar lymphadenopathy makes it less likely than the sarcoidosis hypothesis.

Without repeat gastric biopsy following gluten withdrawal or tissue confirmation of sarcoidosis, a definitive diagnosis cannot be established. The improvement in gastrointestinal symptoms on a gluten-free diet, while encouraging, does not necessarily indicate that the granulomas resolved, as symptom improvement could reflect healing of the duodenal mucosa alone. Furthermore,

sarcoidosis could not be definitively established because no tissue sampling of the thoracic lymphadenopathy or other involved organ was performed, and the patient did not undergo further systemic evaluation.

Limitations

This case has several limitations. First, no repeat gastric biopsy was performed after gluten-free diet initiation, so the granulomatous response to gluten withdrawal could not be assessed. Second, sarcoidosis was not definitively diagnosed, as no tissue sampling of the hilar lymphadenopathy or other involved organ was obtained, and the patient declined further evaluation. Third, infectious causes could not be completely excluded because the recommended special stains (Ziehl-Neelsen and PAS) were not performed. Finally, this is a single observation, and causality cannot be established.

Conclusion

This case highlights the diagnostic challenge of non-necrotizing gastric granulomas in a patient with biopsy-proven Marsh 3C celiac disease and persistent hilar lymphadenopathy, raising the possibility of gastric sarcoidosis. Definitive diagnosis could not be established due to the patient's decision not to pursue further investigation. The case also raises the possibility of an association between celiac disease and systemic granulomatous disease, though causality cannot be inferred from a single observation. Recognition of gastric granulomas in patients with celiac disease should prompt evaluation for systemic granulomatous disorders, particularly when thoracic lymphadenopathy is present**.**

Declarations

Ethics approval and consent to participate: Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Consent for publication: The patient provided written informed consent for the publication of this case report.

Availability of data and materials: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests.

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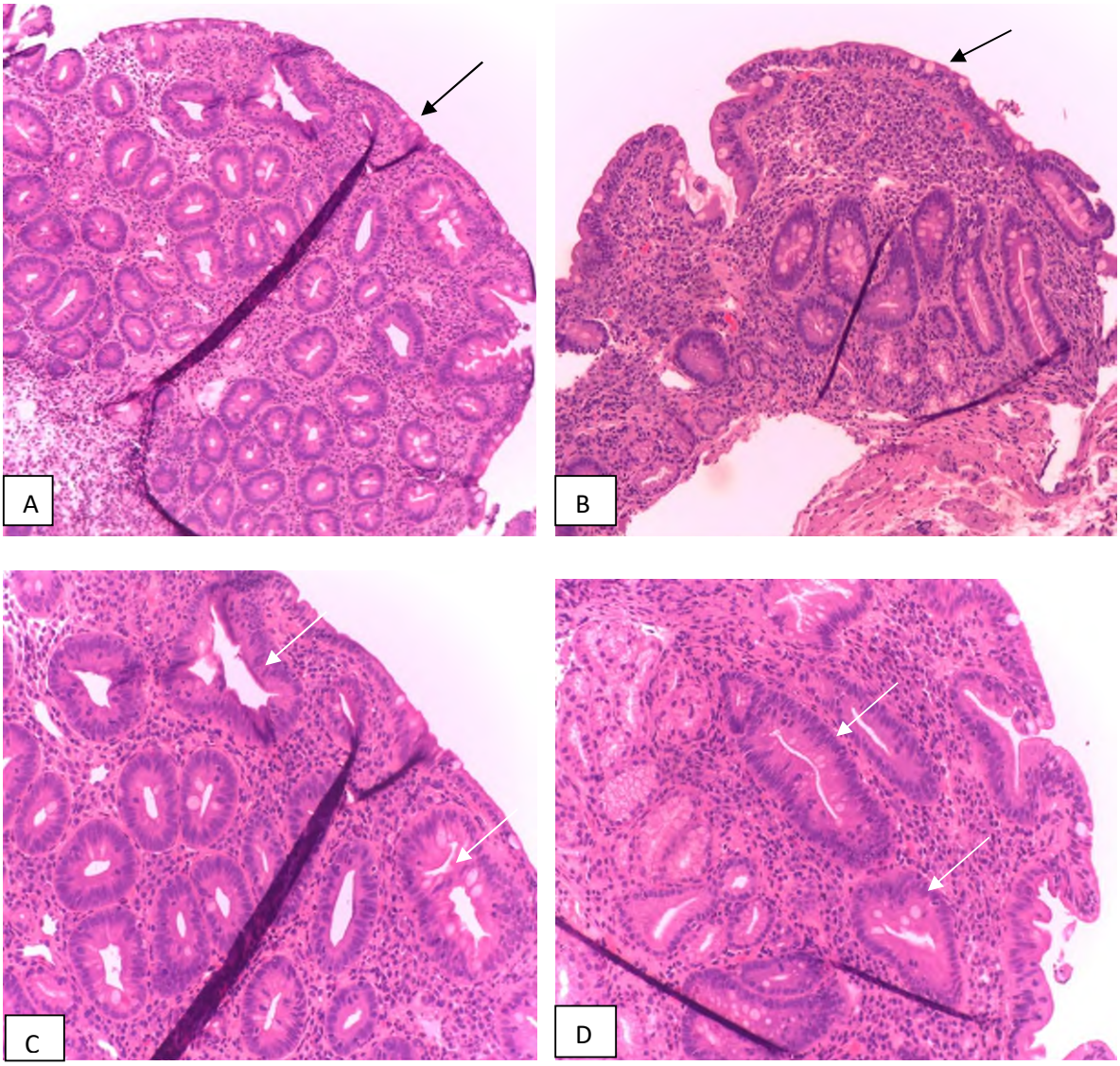
Authors' contributions: All authors contributed equally to this work.

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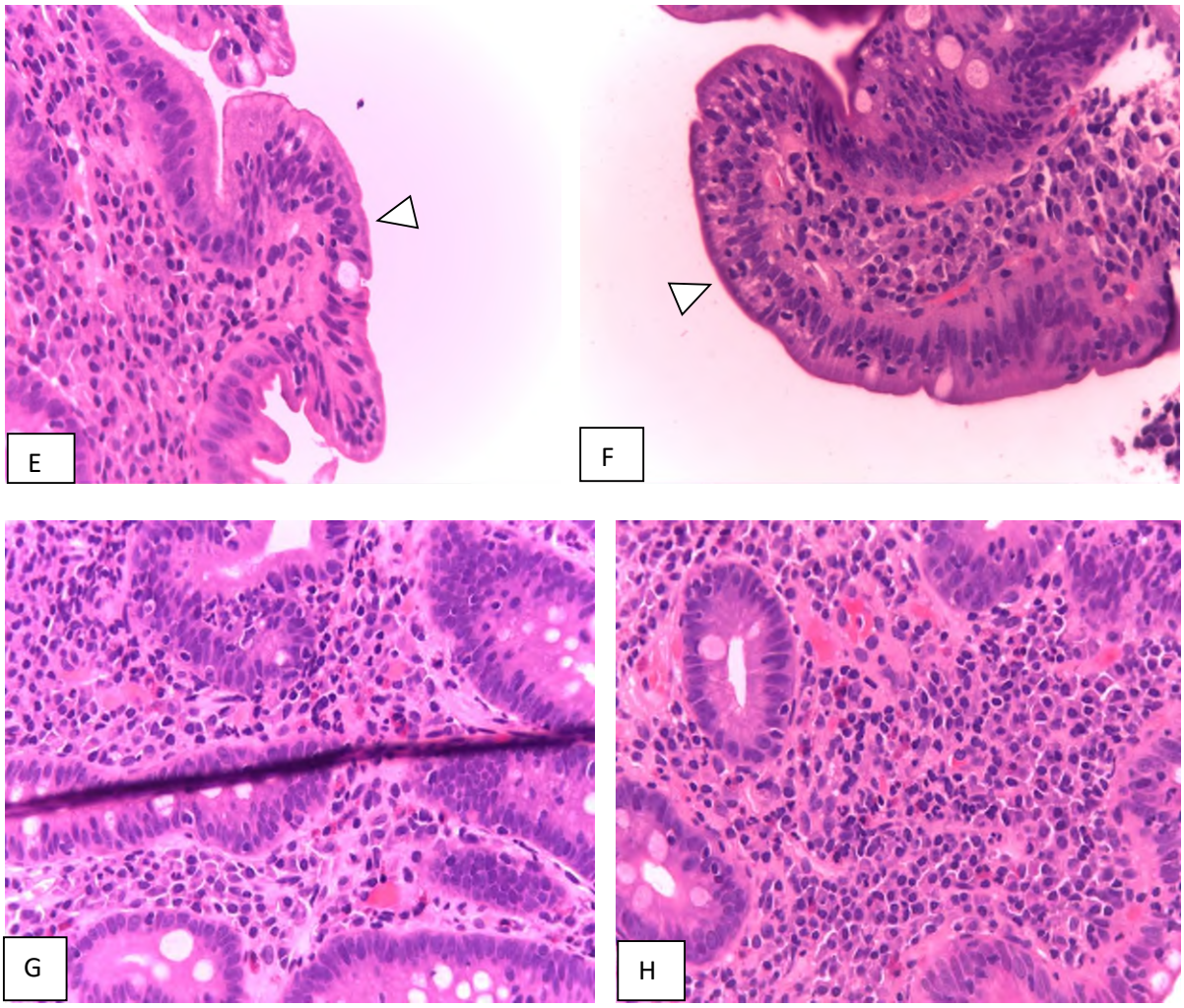


Figure 1: Photomicrographs show celiac disease Histologic features; including total villous atrophy-black arrows (A&B: H&E x10), crypt hyperplasia- white arrows (C&D: H&E x20), and increase intraepithelial lymphocytes; more than 30 lymphocyte /100 enterocytes- black arrowheads (E&F: H&E x40). The lamina propria shows lymphoplasmacytic cell infiltrates in addition to focal neutrophils and eosinophils up to 20 eosinophils/HPF (G&H: H&E x40).

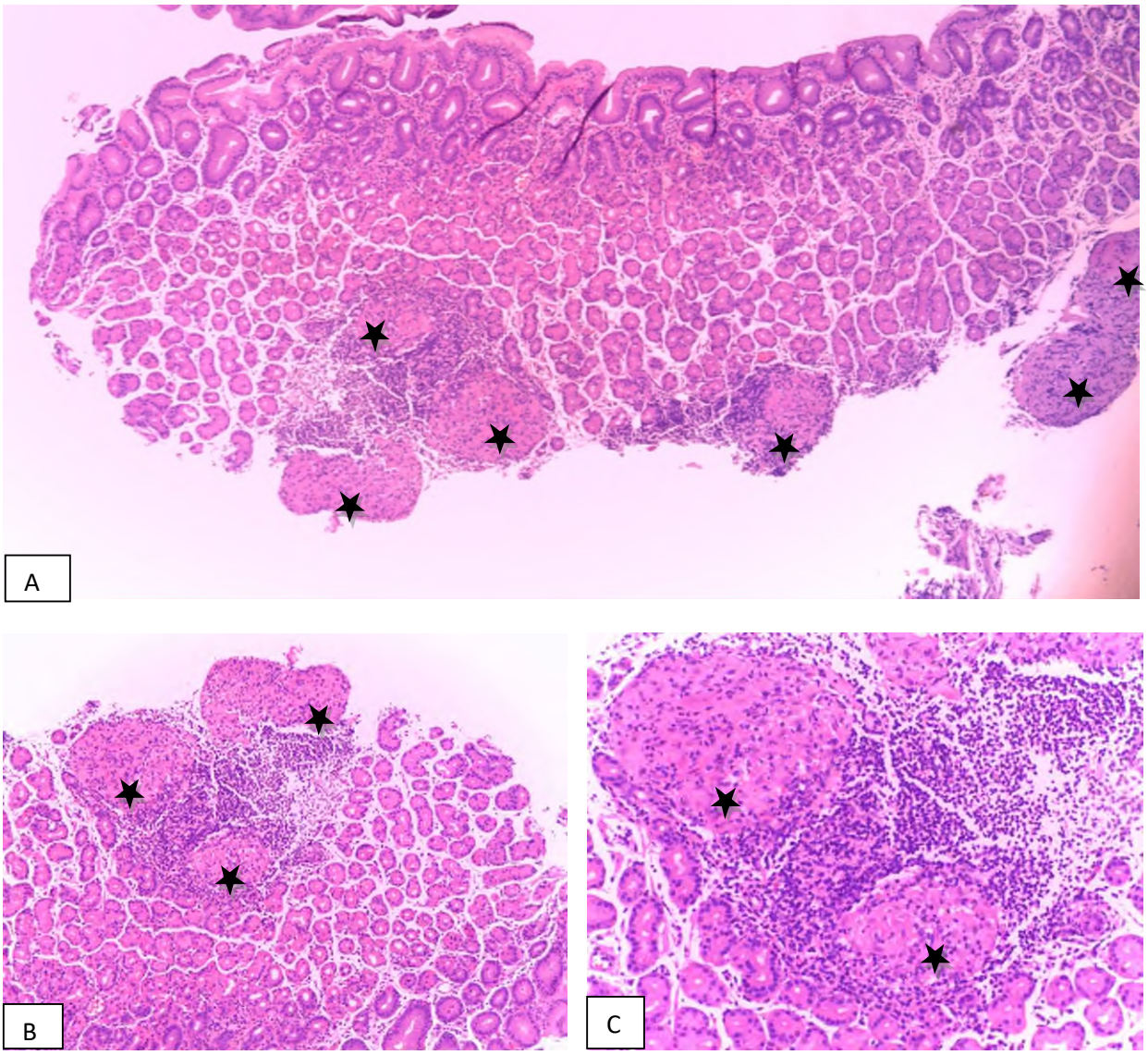


Figure 2: Photomicrographs show gastric mucosa exhibiting non-caseating granulomas- black stars (A, B, C: H&E x4, x10, x20, respectively).

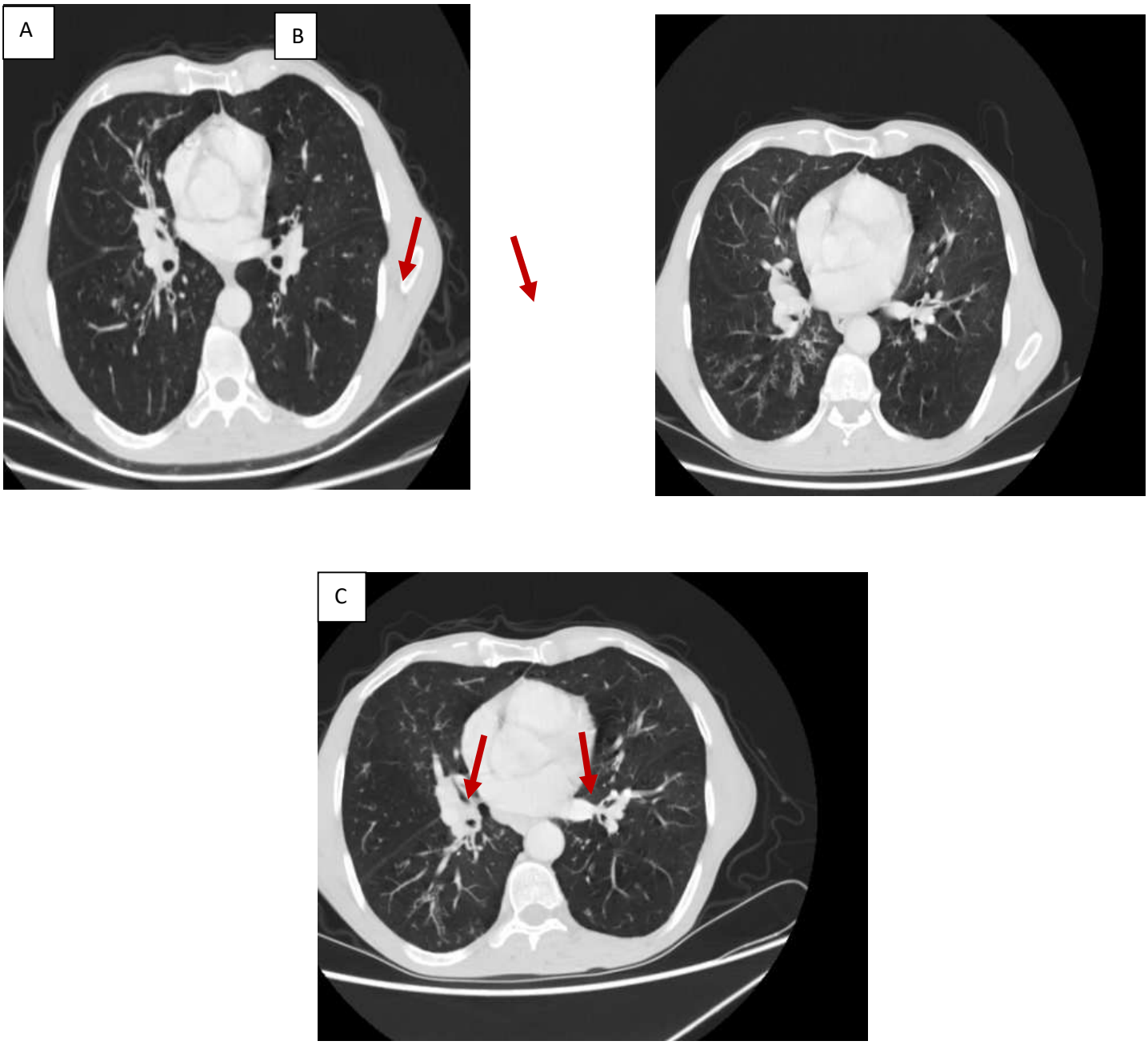


Figure 3: Serial chest CT examinations demonstrating persistent bilateral hilar lymphadenopathy. (A) CT from 2022, (B) CT from 2025, and (C) CT from May 2026. Arrows indicate persistent hilar lymphadenopathy across all three time points

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