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Case report

Nodular Lymphoid Hyperplasia, An Uncommon Benign Entity Associated with Gastrointestinal Bleeding: Report of Two Cases

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Abstract

Introduction: Nodular lymphoid hyperplasia (NLH) is a benign condition characterized by diffuse hyperplasia of lymphoid follicles in the gastrointestinal tract, predominantly in the small intestine. In adults, it is rare, with an unknown sex distribution and a broad clinical spectrum ranging from asymptomatic presentation to nonspecific symptoms such as abdominal pain, flatulence, chronic diarrhea, malabsorption with weight loss, and even gastrointestinal bleeding. Diagnosis is based on characteristic endoscopic findings showing innumerable 2–10 mm nodules, along with histologic confirmation. NLH carries a potential risk for malignant transformation, and its optimal management remains uncertain. **Case presentation:** We describe two male patients in their second and third decades of life who presented with gastrointestinal bleeding and were ultimately diagnosed with NLH following endoscopic and histopathological evaluation. Notably, one patient had a significant surgical history, including appendectomy followed by right colectomy with end-to-end anastomosis for an appendiceal mass during childhood. The second patient presented with coexisting medication-induced liver injury. Both were managed conservatively. **Conclusions:** NLH should be considered in the differential diagnosis of gastrointestinal bleeding given its variable clinical spectrum. Its presence often reflects an underlying condition, most commonly related to immune system disorders. NLH is generally an incidental finding, and management focuses on treating the associated pathology while maintaining vigilant follow-up due to its potential for malignant transformation and the lack of standardized guidance regarding management and surveillance.

Keywords

Benign neoplasms, gastrointestinal hemorrhage, endoscopy, diagnosis, lymphoid tissue, case reports.

INTRODUCTION

Nodular lymphoid hyperplasia (NLH) is a rare, benign condition characterized by the presence of multiple small nodules, measuring between 2 and 10 mm in diameter, with diffuse hyperplasia of lymphoid follicles in the lamina propria and submucosa of the gastrointestinal tract[1]. It mainly involves the small intestine and is less commonly found in the colon and stomach[1,2]. The exact etiology is unknown; however, associations have been described with certain conditions such as immunoglobulin deficiency syndromes, *Helicobacter pylori* (*H. pylori*) infection, giardiasis, human immunodeficiency virus (HIV) infection, and celiac disease[2–5]. Its incidence and sex distribution are unknown. It can occur at any age, although it is more common in children[6]. Two types have been described—focal and diffuse—with the diffuse form being the most common. Clinical presentation is variable, ranging from asymptomatic cases to nonspecific

symptoms such as abdominal pain and flatulence, as well as chronic diarrhea, malabsorption syndromes with weight loss, and even gastrointestinal bleeding[2].

Diagnosis is established based on the classic endoscopic findings of innumerable nodules measuring 2–10 mm, along with histological confirmation showing hyperplastic B-cell follicles in the mucosa and submucosa with mitotically active germinal centers and well-defined lymphocytic mantles. These follicles are cytologically polymorphic and demonstrate lymphocytic polyclonality, confirmed by immunohistochemical or molecular analysis[2,6]. There is no specific treatment, and management is directed toward the associated condition[6]. Given the high risk of extraintestinal and intestinal lymphomas[1,7], NLH represents a risk factor for both intestinal and extraintestinal lymphoma, making diagnosis and follow-up crucial[1]. In adults, NLH infrequently causes gastrointestinal bleeding; the main presentations described include massive obscure bleeding[8,9], recurrent bleeding[10,11], and hematochezia[12]. It has been reported predominantly in immunocompromised patients and less frequently in immunocompetent individuals, and in a few cases no association with an underlying condition has been identified[6]. No association with post-surgical states has been established. Consequently, significant gaps remain in the understanding of this entity. We present two cases and discuss relevant clinical findings, which included gastrointestinal bleeding as the presenting manifestation, in which potential related stimuli—HIV, giardiasis, *H. pylori* infection, and celiac disease—were ruled out, as well as immunocompromise. One case had a history of prior ileocolonic surgery. These represent uncommon associations based on what has been reported to date.

CASE PRESENTATION

Case 1 Presentation

A 24-year-old male patient was admitted with a six-day history of episodes of hematochezia, followed by the onset of melena and epigastric pain, associated with asthenia, adynamia, and nausea. His medical history was notable for appendectomy at eight years of age, with subsequent right hemicolectomy and end-to-end anastomosis due to an appendiceal phlegmon in the setting of complicated acute appendicitis. On admission, he exhibited signs of low cardiac output, including mucocutaneous pallor, a heart rate of 128 beats/minute, and hypotension, with no other relevant findings. Vasopressor support was required, and acute anemia was documented, necessitating transfusional support (**Table 1**). Upper gastrointestinal endoscopy was performed, revealing erythematous corpoantral gastropathy and duodenal ulcers covered with fibrin. Colonoscopy was also performed and showed findings suggestive of nodular lymphoid hyperplasia (NLH) (**Figure 1**). Given these findings and the patient's young age, it was considered appropriate to exclude the presence of lesions in other segments of the small intestine; therefore, antegrade enteroscopy was performed, with no lesions identified in the proximal jejunum.

Table 1. Laboratory Studies Performed during Hospitalization and Outpatient Follow-up

Test	Admission	Follow-up at 41 Days
Leukocytes	4800/µL	4280/µL
Hemoglobin	9.1 g/dL	13 g/dL
Hematocrit	26%	39%
Platelets	250,000 µL	356,000 µL
Acute-phase reactants		
- CRP	1 mg/L	
Coagulation		
- PT	12 seconds	
- INR	1,03	
- PTT	28 seconds	
Renal function		
- Creatinine	1,07 g/dL	
Liver function		
- AST	13 U/L	
- ALT	18 U/L	
- Direct bilirubin	0.14 mg/dL	
- Indirect bilirubin	0.63 mg/dL	
- Total bilirubin	0.77 mg/dL	
Electrolytes		
- Sodium	141 mmol/L	
- Potassium	4.28 mmol/L	
Infectious studies		
- Hepatitis B	Negative	
- Hepatitis C	0.08	
- VDRL	Non-reactive	
- HIV	Negative	

ALT: alanine aminotransferase; AST: aspartate aminotransferase; INR: international normalized ratio; CRP: C-reactive protein; PT: prothrombin time; PTT: partial thromboplastin time; VDRL: Venereal Disease Research Laboratory; HIV: human immunodeficiency virus. Author's own research.

Because NLH was identified in the context of gastrointestinal bleeding, the question arises as to whether this represents the cause of bleeding or an incidental finding. In this case, bleeding was secondary to duodenal ulcers, whereas NLH was found in the colon; therefore, its relationship to the hemorrhagic event is not conclusive.

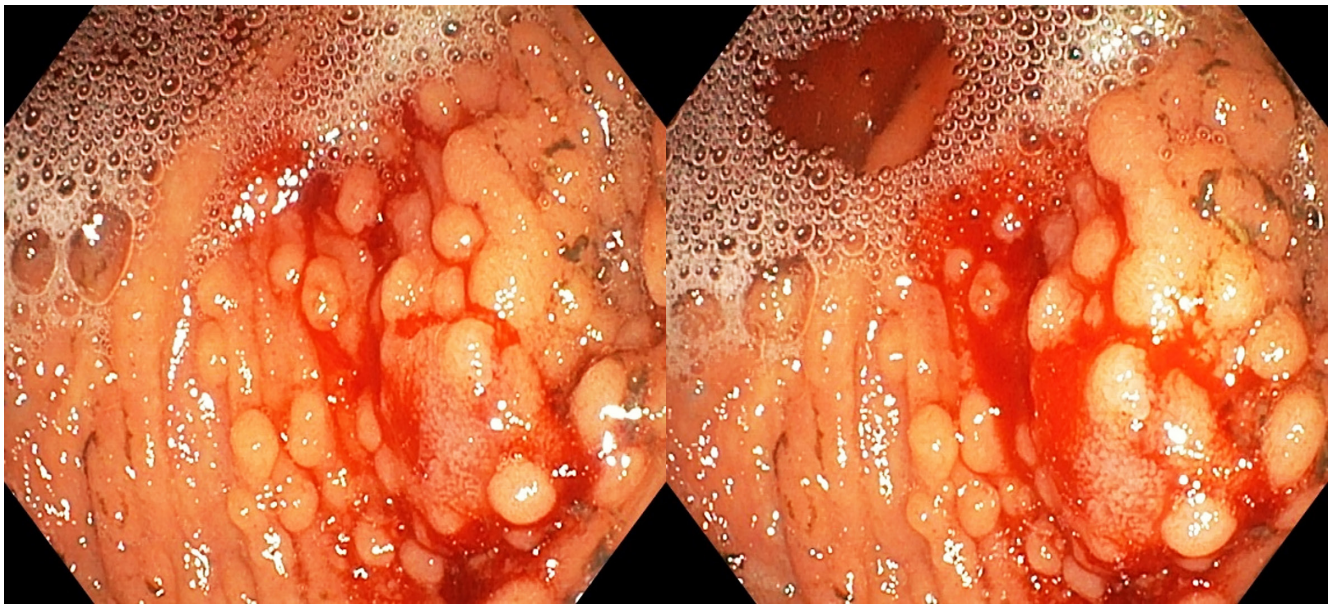


Figure 1. Colonoscopy showing multiple nodular and polypoid lesions, more than 30 in number, involving 100% of the circumference, with diameters ranging from 2 to 10 mm and an extension of approximately 8 cm at the level of the enterocolonic anastomotic loop. Biopsies were obtained. Author's file.

Regarding the patient's immune status, this possibility was explored because NLH has been described in immunocompromised patients. However, a history of appendectomy with right hemicolectomy does not necessarily imply immunosuppression. Although appendectomy may be associated with alterations in intestinal mucosal immunity due to the loss of lymphoid tissue of the appendix, this does not categorize all appendectomized patients as immunocompromised. In this case, it was considered appropriate to evaluate other possible causes of immunosuppression.

The patient subsequently showed an optimal clinical course, with no further need for transfusional support. He was discharged after five days and, at discharge, received empirical therapy for *H. pylori*, despite the absence of histological documentation, given the clinical context. Nevertheless, it was considered necessary to exclude other possible causes of duodenal ulcers before initiating this treatment.

At 41 days, he was evaluated on an outpatient basis, with no recurrence of gastrointestinal bleeding or anemia, and a histopathological report was obtained concluding reactive NLH (**Figure 2**). Given the presentation of this disease in immunocompromised patients, an evaluation for acquired or congenital etiologies was performed, with negative results for HIV infection and hepatitis B and C viruses. In addition, immunoglobulin levels were requested and are pending. To date, the patient has not presented recurrence of symptoms during six months of outpatient follow-up.

Case 2 Presentation

A 19-year-old patient was admitted to the emergency department with a four-month history of episodes of abdominal colic associated with abdominal distension and, occasionally, episodes of hematemesis and hematochezia. There was no relevant past medical history. On admission, vital signs were normal, and no significant findings were observed on physical examination. Laboratory tests showed no anemia and no abnormalities in the hepatic profile (**Table 2**).

Initial studies included an upper gastrointestinal endoscopy performed at admission, which demonstrated erythematous antral gastritis, with no findings suggestive of NLH, as well as a contrast-enhanced abdominal computed tomography (CT) scan without pathological findings. Given the persistence of abdominal pain, lack of therapeutic response to opioid analgesia, and inconclusive initial studies, an exploratory laparotomy was performed, with no evidence of surgical pathology to explain the pain.

Four days after the initial endoscopy, further endoscopic evaluation was undertaken. The second upper gastrointestinal endoscopy identified nodular-appearing mucosa at the level of the gastric antrum, with marked extrinsic compression of the posterior wall of the gastric body, a finding that had not been reported on the initial endoscopic study or the initial CT scan. Additionally, colonoscopy revealed findings suggestive of NLH in the distal ileum and right

colon, prompting biopsy sampling (**Figure 3**). Given the findings on upper gastrointestinal endoscopy, endoscopic ultrasound was performed, which identified gastric compression, possibly secondary to hepatomegaly.

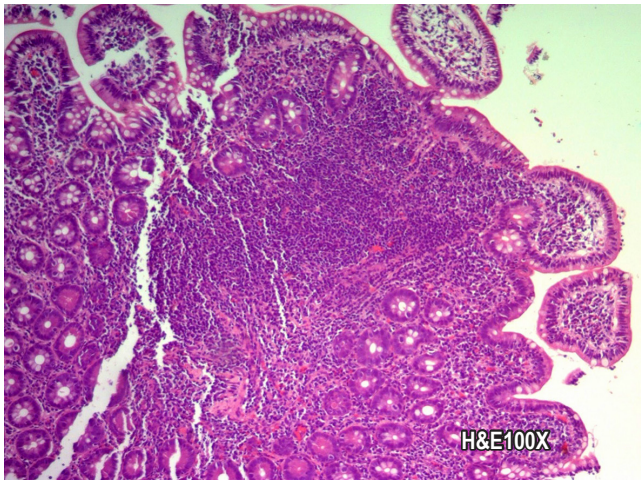


Figure 2. Ileal mucosa with dense lymphoid infiltrate in the lamina propria forming lymphoid follicles with active germinal centers. The epithelium shows mild villous atrophy with an increased number of intraepithelial lymphocytes. Author's file.

During the clinical course, elevated transaminase levels were identified, with predominance of alanine aminotransferase (ALT) and an R ratio of 4.8 (mixed pattern), associated with hepatomegaly on ultrasonography. Given the suspicion of hepatitis, studies for viral, autoimmune, metabolic, and infiltrative etiologies were ordered (**Table 2**), all of which were negative. A liver biopsy was performed, revealing foci of hemorrhagic necrosis and ischemia with minimal acute inflammation. Autoimmune hepatitis was initially considered; therefore, treatment with prednisolone 40 mg/day was initiated and serological studies were expanded (**Table 2**). Once the autoimmune workup was confirmed to be negative, this diagnosis was ruled out, corticosteroid therapy was tapered, and the clinical picture was considered more likely to correspond to drug-induced liver injury, specifically attributed to prolonged exposure to opioids (tramadol).

Subsequently, the patient continued to experience intermittent abdominal pain. A repeat contrast-enhanced abdominal CT scan demonstrated ileal stenosis, a finding that had not been visualized on the prior study. Repetition of the CT scan was deemed necessary to assess the evolution of the clinical course in the setting of persistent symptoms; subsequently, magnetic resonance enterography was performed, with no areas of obstruction identified. Given the unclear etiology of the abdominal pain and the absence of lesions on endoscopic studies, magnetic resonance entero-

Table 2. Laboratory Studies Performed during Hospitalization

Test	Results
Leukocytes	4,200/mm^3
Hemoglobin	15 g/dL
Hematocrit	44%
Platelets	196,000 µL
Acute-phase reactants	
- CRP	0.6 mg/dL
Coagulation	
- PT	11.9 s
- INR	1.06
- PTT	36.6 s
Renal function	
- Creatinine	0.9 mg/dL
- BUN	12 mg/dL
Liver function	
- AST	58 U/L
- ALT	129 U/L
- Total bilirubin	0.2 mg/dL
- Direct bilirubin	0.0 mg/dL
- Indirect bilirubin	0.2 mg/dL
- LDH	147 U/L
Electrolytes	
- Sodium	136 mEq/L
- Potassium	4.3 mEq/L
- Chloride	102 mEq/L
Infectious	
- Hepatitis B	0.68
- Hepatitis C	0.08
- Hepatitis A	15
- VDRL	Non-reactive
- HIV	Negative
Non-infectious	
- Ceruloplasmin	11.5 mg/mL
- Ferritin	119 ng/mL
- Transferrin saturation	41%
- Total iron	148 µg/dL
- Copper	70 µg/mL
- Fecal calprotectin	119 ng/mL
- Urinary porphobilinogen	0.5 mg/24 h
Autoimmunity	
- Immunoglobulin G	9895 mg/L
- Antinuclear antibodies	Negative
- Smooth muscle antibodies	Negative
- Extractable nuclear antibodies	Negative
- Anti-liver/kidney microsomal antibodies	Negative

ALT: alanine aminotransferase; AST: aspartate aminotransferase; BUN: blood urea nitrogen; INR: international normalized ratio; LDH: lactate dehydrogenase; CRP: C-reactive protein; PT: prothrombin time; PTT: partial thromboplastin time; VDRL: Venereal Disease Research Laboratory; HIV: human immunodeficiency virus. Author's own research.

graphy, and CT imaging, video capsule endoscopy (VCE) was indicated (**Figure 4**). This study ruled out involvement of the stomach, duodenum, and jejunum and identified NLH limited to the ileum (**Figure 5**) in a patient without immunocompromise and without association with infectious processes.

The patient demonstrated an adequate clinical course and was discharged after 10 days, without recurrence of

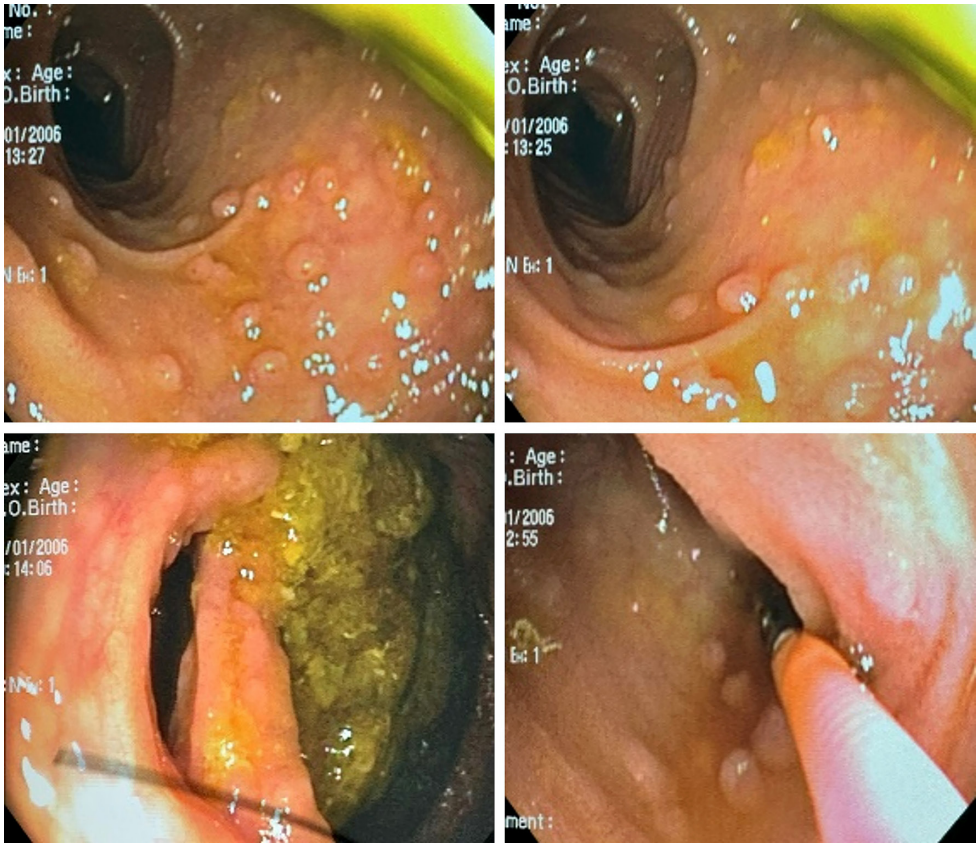


Figure 3. Colonoscopy showing multiple nodular lesions, more than 30 in number, involving 100% of the circumference, with diameters ranging from 2 to 10 mm and an extension of approximately 8 cm at the level of the distal ileum, ileocecal valve, cecum, and ascending colon. Biopsies were obtained. Author's file.

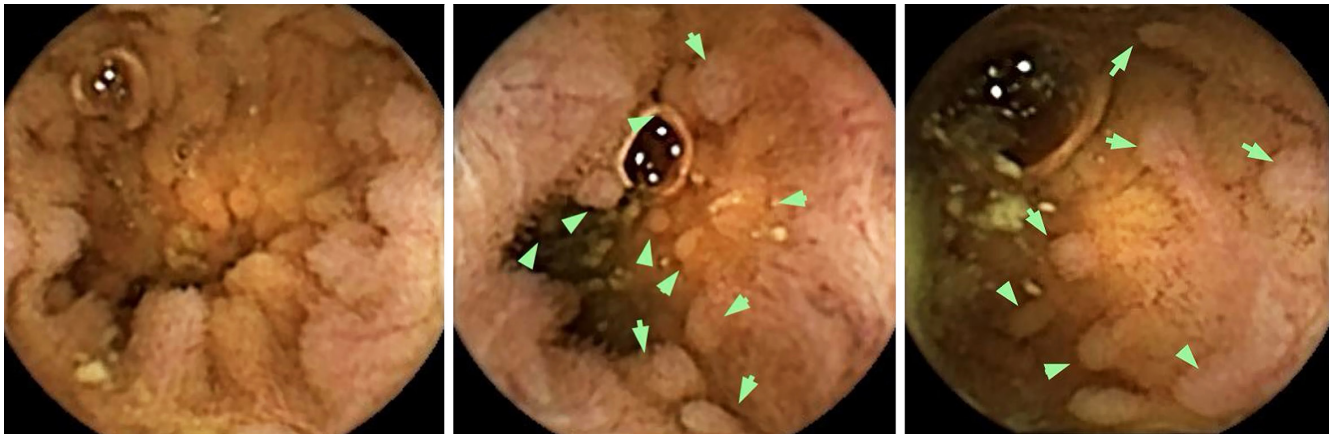


Figure 4. Video capsule endoscopy showing multiple nodular lesions with complete circumferential involvement, predominantly in the distal ileum, ileocecal valve, cecum, and ascending colon. Author's file.

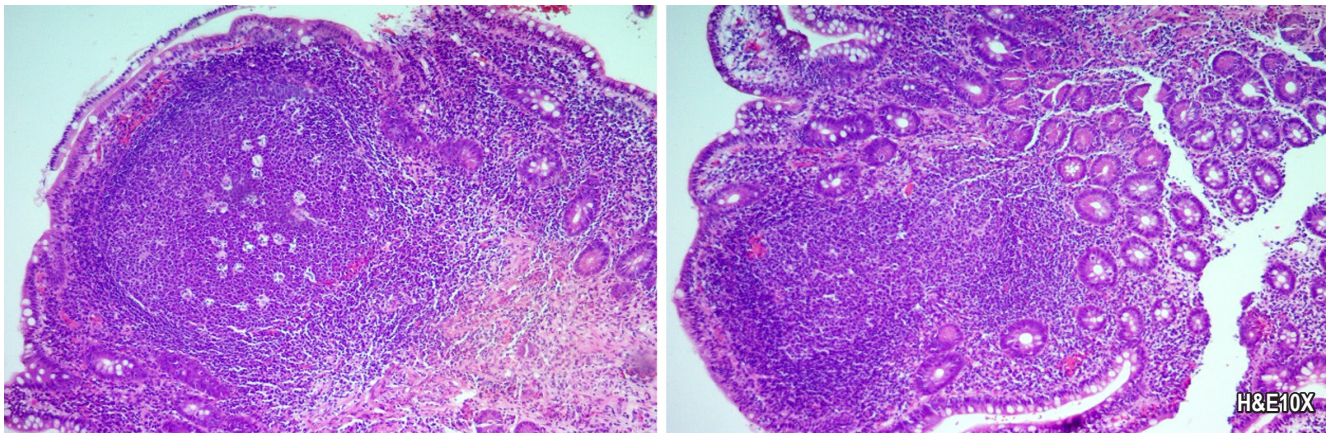


Figure 5. Ileal biopsy showing evidence of chronic inflammatory infiltrate, presence of lymphoid follicles with reactive germinal centers, and increased lymphocytic infiltrates within the villi, findings consistent with reactive nodular lymphoid hyperplasia. Author's file.

symptoms. Outpatient follow-up was scheduled for six months later.

DISCUSSION

Several features of these cases are noteworthy and warrant analysis in light of current knowledge. One of the characteristics that make these cases unique is that they involved young adults presenting with gastrointestinal bleeding. In one patient, there was a prior surgical history involving the ileum and proximal colon, in addition to the age at presentation and the absence of associated secondary pathology. Conversely, some typical features were also observed, including documentation of lesions predominantly involving the ileum and, in one case, achievement of clinical stability without recurrence of gastrointestinal bleeding following empirical therapy for *H. pylori*.

The proposed classification of NLH includes diffuse NLH and focal forms, which mainly affect the terminal ileum, rectum, or other areas of the digestive tract[13–15]. In adults, management is guided by the presence or absence of immunodeficiency[16], although NLH is usually associated with immunodeficiency and giardiasis[3]. In states of immunodeficiency, its presentation has been proposed to result from a compensatory state of intestinal lymphoid tissue with inadequate function, leading to the accumulation of plasma cell precursors due to defects in B-cell maturation and development[17]. Conversely, in the absence of immunodeficiency, NLH has been proposed to arise from immune stimulation of intestinal lymphoid tissue by antigenic triggers, possibly infectious, resulting in repetitive stimulation and eventual hyperplasia of lymphoid follicles[18]. Of the cases described, one corresponded to focal NLH with ileal involvement and absence of immunodeficiency; the other more likely represented diffuse NLH with gastric and ileal involvement, in which the main infectious agents associated with NLH were ruled out. This raises several hypotheses regarding prior noxious exposures related to the presentation in both cases: in one, ileal and proximal colon resection with end-to-end anastomosis; in the other, the inability to exclude unidentified infectious insults or antigenic stimulation during early life. Nor could the hypothesis proposed by Chiaramonte and colleagues[7] be excluded, in which NLH represents a transitional stage in the development of a malignant lesion or, possibly, an early-stage lymphoma.

The spectrum of entities associated with NLH is broad and includes selective immunoglobulin A (IgA) deficiency, celiac disease, common variable immunodeficiency, HIV infection, *H. pylori* infection, and giardiasis[3]. Many of these conditions have been described in both immunocompromised and immunocompetent patients; therefore, in the initial diagnostic approach, investigations are directed toward these etiologies. Conditions related to immunodeficiency have been described predominantly with diffuse involvement[6], showing classic endoscopic findings in the jejunum, ileum, and rectum, whereas giardiasis and *H. pylori* infection have been mainly associated with classic endoscopic findings in the stomach, duodenum, and proximal jejunum[3,6,19]. An association between NLH and non-exophytic colonic neoplasms, adenomas, and adenocarcinomas has also been suggested, with these entities showing classic endoscopic findings particularly in the terminal ileum[20]. It should also be noted that in other cases no association with any of these entities has been described, and the clinical and endoscopic behavior in such cases is even less well understood[21].

In both cases described, classic endoscopic findings were predominantly identified in the ileum; therefore, extended endoscopic and imaging studies were performed due to suspicion of neoplasia or adenomas in locations not assessable by initial endoscopic methods. It should also be noted that the decision to initiate empirical therapy for *H. pylori* in one of the described cases was supported by endoscopic findings of duodenal ulcers in the context of gastrointestinal bleeding, rather than by suspicion of NLH.

Both cases presented with gastrointestinal bleeding at onset. This clinical manifestation is unusual in the literature to date regarding NLH; moreover, in adults, this form of presentation is less well characterized than in the pediatric population[6,8,11]. Among bleeding manifestations, the best-described in adult NLH is hematochezia, followed to a lesser extent by obscure-origin bleeding and massive bleeding[9,22]. One of the patients described had duodenal ulcers with signs of recent bleeding, which was associated with greater severity of the bleeding episode requiring transfusional support. In both cases, ileal involvement by NLH was documented by colonoscopy with distal ileoscopy and by VCE.

Common causes of mid-gastrointestinal bleeding include angiodysplasias, intestinal tumors, ulcers, erosions, and entero-vascular fistulas[23]. In addition, NLH should be considered as a differential diagnosis after these entities in the context of overt obscure gastrointestinal bleeding in adults[22]. Definitive management in the setting of gastrointestinal bleeding remains highly debated in the literature to date and depends on the severity of the clinical presentation. Greater experience exists in pediatric patients, with therapeutic options ranging from conservative medical management to more radical approaches, including surgical or interventional treatment, the choice of which varies on a case-by-case basis[8,9]. In both cases, supportive care and clinical surveillance with symptomatic management were provided, with no other associated conditions identified. Treatment is directed toward management of associated conditions, as this disorder alone generally does not require intervention[24]. Surgical management represents a controversial measure and may result in unnecessary radical resections due to diagnostic difficulty[2].

In the cases described, periodic outpatient follow-up was considered and, depending on clinical evolution, additional imaging or endoscopic studies were to be considered. Currently, there is no consensus regarding follow-up in NLH; however, surveillance is recommended given the risk of malignant transformation[2]. Regarding the preferred modality for follow-up, imaging studies such as computed tomography enterography or barium contrast series may be useful; however, these modalities have limitations for the early detection of precancerous or cancerous lesions[25]. VCE and double-balloon enteroscopy represent excellent options for follow-up in NLH and are particularly useful for ruling out complications and determining disease extent[26]. However, they are not formally indicated for diagnosis or routine follow-up, and when considered, their limitations must be taken into account, including availability, cost, and the need for experienced personnel to perform them[2]. The duration and interval of follow-up have also not been defined. Consequently, follow-up in NLH represents a challenge in clinical practice due to the scarcity of recommendations or guidelines, limited information regarding its association with malignant conditions, and the lack of a single preferred modality. Therefore, decisions regarding follow-up should be guided by multiple factors, including age, the presence of alarm signs and symptoms, the type of NLH involvement documented, the presence of related secondary pathology, and associated immunocompromise.

CONCLUSIONS

NLH is a benign condition, infrequent in adults, with a variable clinical spectrum, whose presence usually indicates an underlying cause, most often related to immune system disorders. Management is focused on treatment of the associated condition, and NLH has been associated with malignant transformation. It represents a challenge in clinical practice, given the difficulty of diagnosis—which must be supported by endoscopic and histopathological findings—the limited information available on multiple aspects of the disease, and the absence of guidelines for standardized management and follow-up. Therefore, definitive decisions regarding treatment and follow-up should be guided by multiple factors that vary on a case-by-case basis.

DECLARATIONS

Ethical Approval and Participant Consent

This report was approved by the patient and their family.

Consent for Publication

Written informed consent was obtained from the patient for the publication of this case report and the accompanying images.

Data and Material Availability

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Conflict of Interests

The authors declared no competing interests.

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Author Contributions

All authors analyzed and interpreted the patient data, wrote the manuscript, analyzed and interpreted the images, and contributed to manuscript preparation. All authors read and approved the final manuscript.

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