Localized Lymphoid Hyperplasia of the Colon Mimicking Lymphoma in a Patient with Ulcerative Colitis

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Abstract

Localized lymphoid hyperplasia (LLH) of the colon is a rare clinical entity often considered benign, typically resolving without intervention. However, its endoscopic appearance may resemble colonic lymphoma, such as MALT-lymphoma or diffuse large B-cell lymphoma, underscoring the importance of histopathological evaluation for accurate diagnosis. Here, we present a case of a 54-year-old asymptomatic man with ulcerative colitis (UC) diagnosed with LLH of the rectum. While rectal bleeding is the most common presentation of LLH, patients can be asymptomatic. Although rare, colonic lymphoma has been reported in patients with UC. Clinicians should refrain from diagnosing lymphoma without histopathological confirmation and recognize the typically benign course of LLH in most cases.

Keywords: Lymphoma, rectal polyp, ulcerative colitis

INTRODUCTION

Localized lymphoid hyperplasia (LLH) of the colon, also referred to as rectal tonsil, lymphoid polyp, or benign lymphoid polyp, is a rare endoscopic finding in adults.1 When present, LLH is almost exclusively seen in the rectum, particularly just above the dentate line.1,2 LLH can manifest as a polyp of varying sizes or a nodule/mass. It frequently presents with rectal bleeding and, less commonly, with symptoms such as abdominal pain, chronic diarrhea, or intestinal obstruction.1

The exact cause of LLH is not well understood, although it has been associated with several factors, including Epstein-Barr virus, human immunodeficiency virus (HIV), Helicobacter pylori, giardiasis, common variable immunodeficiency, selective IgA deficiency, and celiac disease.3–5 LLH can occur across all age groups but is more prevalent in pediatric populations compared to adults.3

LLH is typically regarded as a benign lesion, and in the majority of cases, it resolves without the need for any intervention.5,7 However, some reports suggest its association with marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT), which is the second most common type of rectal lymphoma.1,2

Differentiating between LLH and MALT-lymphoma can be challenging and may necessitate in-depth histopathological analysis and genetic evaluations to ensure an accurate diagnosis. Histologically, LLH is characterized by prominent, well-defined lymphoid tissue within the submucosa and overlying lamina propria of the mucosa, as well as markedly hyperplastic germinal centers.4 While the histopathological features of LLH differ from those of MALT-lymphoma, which typically displays monocyctoid B cells, including marginal (centrocyte-like) cells, and small lymphocytes with distortion of the glands and invasion of nearby reactive germinal centers, at low power view, the dense lymphoid infiltrate in LLH may resemble lymphoma, leading to potential diagnostic challenges.2,5,9,10

There is currently no consensus on whether surveillance endoscopies are necessary in cases of LLH, as the majority of patients have been reported to experience no recurrence.7 In this report, we present a case of LLH in a patient with ulcerative colitis (UC).

CASE PRESENTATION

A 54-year-old male with a six-year history of ulcerative colitis (UC) presented to the gastroenterology clinic following a positron emission tomography computed tomography (PET-CT) scan that revealed abnormal metabolic activity in the rectum. He had a past medical history of squamous cell carcinoma of the lung, for which he underwent curative resection surgery 16 years ago. During a subsequent follow-up, the current PET-CT scan was performed, prompting his visit to our clinic. His UC was in remission, and he reported no symptoms. Laboratory tests, including cytomegalovirus viral tests, were unremarkable. His medication regimen included oral mesalamine at a dosage of 4 g/day. He had not required steroids or any immunomodulatory therapy previously. Given the PET-CT findings, a decision was made to perform a colonoscopy.
The colonoscopy revealed a horseshoe-shaped area with ulceration in the middle of the rectum, as shown in Figure 1A. Multiple biopsies were taken, and the histopathological analysis of the lesion, shown in Figure 2, revealed superficial mucosal fragments of lymphoid hyperplasia with active chronic inflammation. Lymphoid proliferation was observed between the lamina propria and submucosa, with 75-80% of the cells being B lymphocytes and 20-25% being T lymphocytes. No cryptitis or abscesses were observed, and Cytokeratin AE1/AE3 immunostaining was negative. Dysplasia was not present, leading to the diagnosis of LLH of the rectum. A follow-up colonoscopy at month 3 showed regression of the lesion, as shown in Figure 1B.

**DISCUSSION**

Despite rectal bleeding being the most common presentation of LLH, patients can be asymptomatic, as seen in our case. Previously, five cases of LLH have been reported in patients with UC during follow-up endoscopies. However, there is no established causation between the two entities. In our case, the patient’s UC was in remission with mesalamine therapy, and he exhibited no active inflammation or clinical symptoms. The strength of this case report is that it provides additional data regarding LLH occurring in UC, indicating a benign course. However, the limitation is the lack of long-term follow-up beyond six months post-resection of the lesion.

Colonic lymphomas are rare, comprising 10-20% of all gastrointestinal lymphomas. Among colorectal non-Hodgkin lymphomas (NHL), diffuse large B-cell lymphoma is the most common histologic subtype, characterized by its aggressive nature, and MALT-lymphoma is the second most common subtype. Although exceedingly rare, cases of MALT-lymphoma occurring in UC have been previously reported. Histopathological analysis plays a crucial role in excluding a malignant process, making an accurate diagnosis, and preventing unnecessary radical management. It is important for clinicians to refrain from making an unwarranted diagnosis of lymphoma before histopathological evaluation and to recognize the benign course of the disease in the majority of patients.

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**REFERENCES**


