

Unusual polypoid mass in the colon

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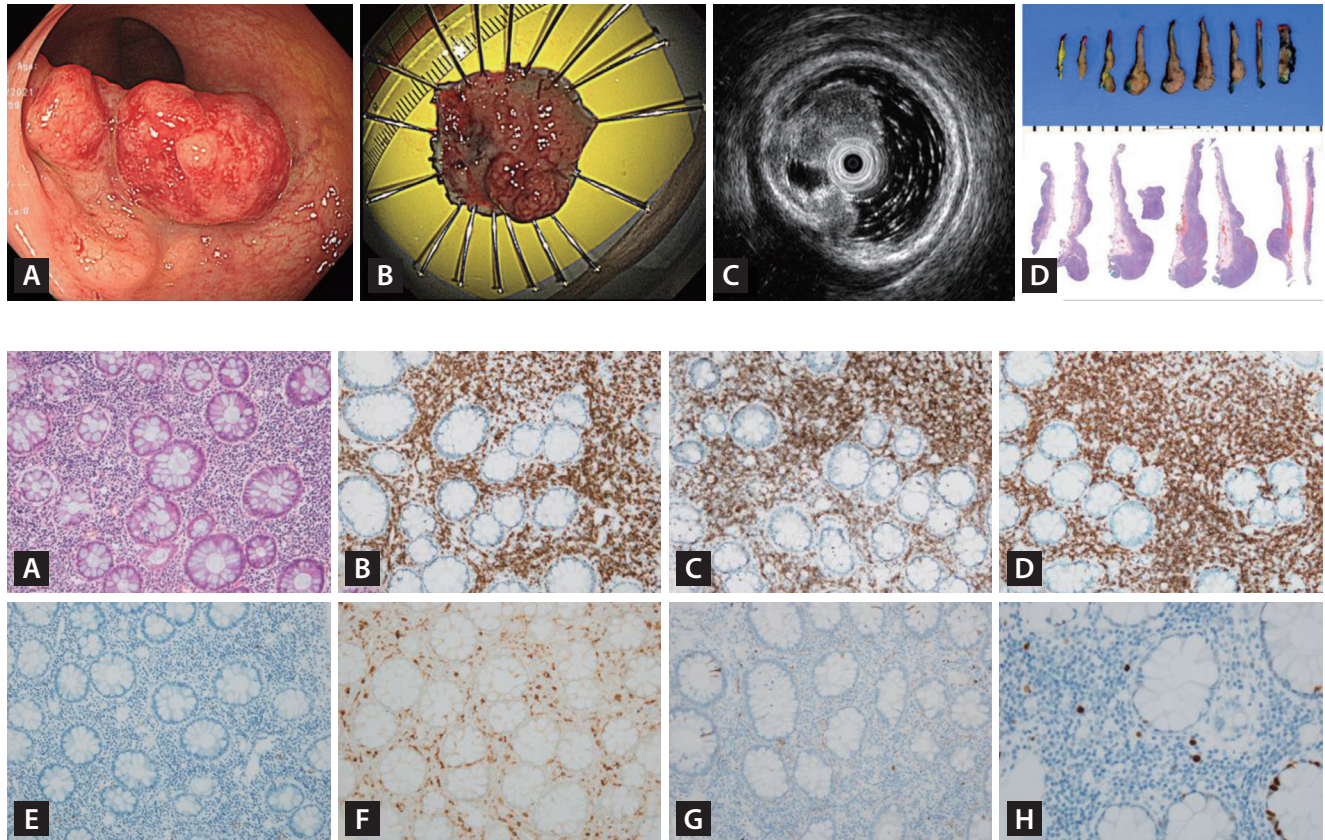


Figure 1. (A) A lobulated polypoid mass measuring 4.5×2.0 cm was observed 20 cm above the anal verge during colonoscopy. (B) A fresh mass was identified in the endoscopic submucosal dissection specimen. (C) Endoscopic ultrasonography revealed a hypoechoic lesion located between the mucosa and submucosa. (D) Top: cut surfaces of the mass fixed in formalin showed a raised, flat, solid mass. Bottom: a microscopic image from scanned glass slides revealed lymphoid cells infiltrating the thickened mucosa (hematoxylin and eosin stain [H&E], $\times 0.4$).

Figure 2. Histopathologic examination. (A) Dense small lymphoid cells infiltrate the expanded lamina propria without significant epitheliotropism. These lymphoid cells are positive for (B) CD3, (C) CD5, and (D) CD8, but negative for (E) CD20, (F) CD4, and (G) CD56. (H) The Ki-67 proliferation index is less than 5% (A: H&E, $\times 200$; B-G: immunohistochemistry, $\times 200$; H: immunohistochemistry, $\times 400$).

A 79-year-old male visited the outpatient department for a screening colonoscopy. A 4.5×2.0 cm lobulated polypoid mass was detected 20 cm above the anal verge in the sigmoid colon (Fig. 1A). The lesion in the colon was a localized mass with suspicion of lymphoma. Due to inconclusive bi-

opsy results, endoscopic submucosal dissection (ESD) was performed for both diagnostic and therapeutic purposes. After admission, ESD was performed (Fig. 1B). Endoscopic ultrasound revealed a hypoechoic lesion located between the mucosal and submucosal layers (Fig. 1C). Pathological-

ly, the specimen showed a thickened mucosa and monomorphic small lymphoid cell infiltration in the expanded lamina propria with nondestructive glands (Fig. 1D, 2A). These lymphoid cells expressed CD3+, CD5+, CD8+, low Ki-67 expression (less than 5%), CD20-, CD79a-, CD4-, CD56-, granzyme B-, CD10-, BCL6-, CD30-, cyclin D1-, and EBV-encoded small RNA by in situ hybridization (Fig. 2B-H). The molecular pathology identified monoclonal T-cell receptor (TCR) β , δ , and γ gene rearrangement, leading to a diagnosis of indolent T-cell lymphoproliferative disorder of gastrointestinal tract (iTLPD-GI). The patient was asymptomatic, and given the successful endoscopic resection and the low morbidity associated with the disease, we opted for follow-up observation without further treatment [1]. The patient remains alive for three years. Initially described as iTLPD-GI, it was reclassified as indolent T-cell lymphoma of the gastrointestinal tract (iTCL-GI) in the 5th edition of the WHO classification [2-4].

During colonoscopy, iTCL-GI may present mucosal thickening, nodularity, polyps, irregular mucosal surfaces, and ulcers [5]. A key diagnostic feature of iTCL-GI is clonal small T cells infiltrating the expanded lamina propria without epitheliotropism. Immunohistochemistry and TCR gene rearrangement analysis are essential for diagnosis. Non-neoplastic diseases and other lymphomas should be excluded. Clinicians and pathologists must recognize this rare entity, and clinicopathological correlations are crucial for an accurate diagnosis.

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Seung-Woo Lee: conceptualization, writing - original draft; Jong Ok Kim: writing - original draft, writing - review & editing

Conflicts of interest

The authors disclose no conflicts.

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Ethical statement

This case was approved by the Institutional Review Board of Daejeon St. Mary Hospital, College of Medicine, Catholic University of Korea (IRB No. DC24ZAS10058).