

T-CELL LYMPHOMA MISDIAGNOSED AS CROHN'S DISEASE: CASE REPORT

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For many years, there has been a concern that inflammatory bowel disease carries an increased lymphoma risk. At the same time, patients with intestinal lymphomas are occasionally misdiagnosed as having Crohn's disease. We report a case of T-cell lymphoma of the bowel misdiagnosed as Crohn's disease, which illustrates the diagnostic challenges posed by peripheral extranodal lymphomas. A 68-year old female presented with clinical symptoms (diarrhoea, abdominal pain, poor appetite and significant weight loss), and colonoscopic and initial histological findings that were similar to inflammatory bowel disease. She was diagnosed with Crohn's disease and received treatment with sulfasalazine with subsequent improvement of symptoms. Eight months after the initial diagnosis the patient experienced sudden abdominal pain. Laparotomy revealed necrosis in the small and large intestine and ileostomy was performed. On day 10 of a complicated postoperative period the patient died. Post-mortem histopathological examination of small and large intestine revealed highly malignant peripheral T-cell lymphoma, not otherwise specified. Differentiation of intestinal T-cell lymphoma from Crohn's disease continues to be a challenge, because clinical, colonoscopic, radiological and histopathological findings can mimic Crohn's disease. Careful multi-disciplinary assessment and knowledge of this rare disorder is crucial for timely diagnosis.

Key words: T-cell lymphoma, PTCL-NOS, Crohn's disease, inflammatory bowel diseases.

INTRODUCTION

Peripheral T-cell lymphomas (PTCL) are post-thymic/mature T-cell haematological tumours that constitute about 15% of all non-Hodgkin's lymphomas in adults. Peripheral T-cell lymphoma, not otherwise specified (PTCL-NOS), is the most common subtype of PTCL in Western countries (Vose *et al.*, 2008). PTCL-NOS corresponds to a heterogeneous group of nodal and extranodal lymphomas that do not fit any of the defined entities of the PTCL group.

PTCL-NOS often presents at an advanced stage, has an aggressive course and a poor outcome (Swerdlow *et al.*, 2017). Patients often have constitutional symptoms, generalised lymphadenopathy and extranodal involvement. If the

gastrointestinal tract (GI) is involved, symptoms of PCTL-NOS may include abdominal pain, fever, bloody stool and chronic diarrhoea (Sun *et al.*, 2011; Chen *et al.*, 2015). The presence of profound anaemia and the hemophagocytic syndrome often suggest the T-cell nature of the tumour (Rodríguez-Abreu *et al.*, 2008). Although PTCL-NOS is a more common type of PTCL, differentiation from other T-cell neoplasms, such as enteropathy-associated lymphoma (EATL), is often needed.

Here we report a case of PTCL-NOS of the gastrointestinal tract misdiagnosed as Crohn's disease (CD), which illustrates the diagnostic challenges posed by peripheral extranodal lymphomas.

CASE DESCRIPTION

In 2012, a 68-year old female was admitted to the emergency room for the first time with a history of nausea, vomiting, watery diarrhoea five times a day and 39 °C fever for two days. There were no abnormal findings. The patient had undergone mastectomy, hysterectomy and ovarian amputation, and chemo- and radiation therapy because of breast cancer in 1994. The patient was discharged with the diagnosis of acute gastroenterocolitis of unknown aetiology.

In March 2014, the patient was hospitalised with complaints of diarrhoea five times a day without blood or mucus, lower abdominal pain associated with eating, poor appetite, sweating and weight loss of 7 kg during the last three months. During hospitalisation, she continued to experience severe abdominal pain, diarrhoea 5–7 times a day with mucus, loss of appetite, weakness and subfebrile temperature. Laboratory testing showed normocytic anaemia.

Initial clinical evaluation and colonoscopy with following histology revealed features similar to CD. On histological examination severe glandular dysplasia was detected (Fig. 1). The patient was prescribed sulfasalazine 1 g TDS and her clinical condition stabilised.

In April, she developed sudden chest pain. ECG and laboratory findings showed acute myocardial infarction and acute coronarography with angioplasty was performed. The patient was discharged in good condition.

In June, she was admitted for a follow-up. She reported passage of loose stools without blood or mucus five times a day. Laboratory findings included normochromic macrocytic anaemia with HGB 11.2 g/dL and CRP > 20.5 mg/L. The patient received mesalazine 800 mg QID for four days and methylprednisolone 16 mg OD for 2 days. CT scan with contrast agent showed colon thickening up to 1.7 cm and colon descendens distal area narrowing (Fig. 2). No liver or lymph node abnormalities were seen, and mild splenomegaly was noted. Follow-up colonoscopy was scheduled for October.

In the beginning of September, she was admitted due to a suspicion of CD relapse. Laboratory findings showed leukopenia (WBC $3.75 \times 10^9/l$), hypochromic normocytic anaemia (RBC $2.47 \times 10^6/mkl$, HGB 8.4 g/dl), hyponatraemia 126 mmol/l, hypocalcaemia 1.95 mmol/l, and hypoproteinaemia with total protein 50.6 g/l. Abdominal ultrasonography showed ascites and moderate splenomegaly. Upper endoscopy was without pathological changes. Colonoscopy revealed the descending colon had circumferential mucosal thickening and irregularity approximately 60 cm proximally to the anal verge; the sigmoid colon had circumferential mucosal thickening and irregularity 30 cm proximally to the anal verge. Appearances were suggestive of a discontinuous (patchy) inflammatory condition affecting the large intestine, most likely CD.

On 28 September, she complained of sudden abdominal pain. Laboratory findings included the following: HGB

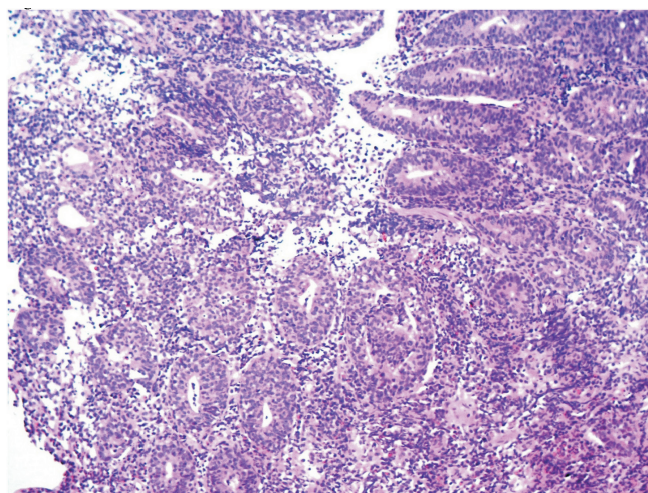


Fig. 1. Ileocecal zone endoscopic biopsy. Haematoxylin eosin, 100x. Marked inflammatory reaction with neutrophils, eosinophils, lymphocytes and macrophages in the lamina propria with high grade dysplasia in crypt epithelium.

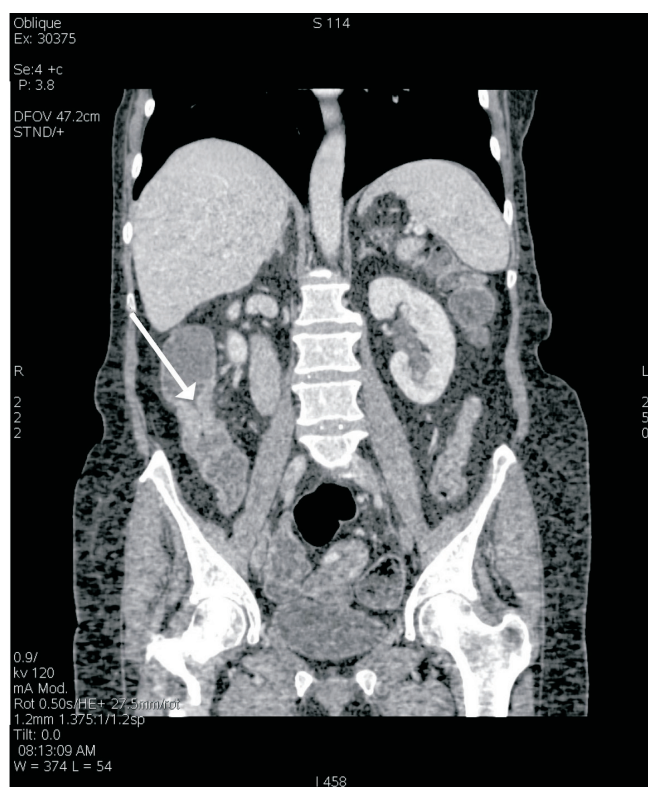


Fig. 2. Abdominal CT scan with contrasting agent. Colon descendens distal area narrowing with bowel wall thickening up to 1.7 cm.

(10.90 g/dl), elevated CRP (189.71 mg/l) and procalcitonin (6.70 ng/ml). CT scan showed intestinal microperforation. Subsequent laparotomy revealed necrosis in the small intestine 25 cm proximally to the ileocecal valve and in the large intestine. Ileocaecal resection material was sent for histological examination, which revealed wall structure changes of the small intestine: totally effaced architectonics, ulceration covered with fibrin, detritus and WBC. There was diffuse transmural infiltration with atypical T-lymphocytes. T-lymphocytes were small to medium sized with regular round or oval nuclei, and were positive for CD3, BCL2,

LCA, focally positive for CD4 and CD8, and negative for CD5, Cyclin D1, CD79a, CD20 and CD30 markers. High proliferative activity was detected in the neoplastic cells (Ki67 labelling index 50–70%).

Histopathological examination of the small intestine revealed highly malignant PTCL-NOS (Figs. 3–7).

Histopathological examination of the colon showed multiple mucosal superficial erosions, high level reactive regeneration of the crypt epithelium with focal mild to severe dysplasia, as well as diffuse infiltration of lymphocytes, neutrophils and eosinophils in the lamina propria, sclerotic changes in the submucosa. Immunohistochemically the epithelium was positive for CKAE AE1/AE3, with Ki67 90% in regenerative crypt epithelium. No granulomas were found in the colon, with no features characteristic of CD or ulcerative colitis. These findings were considered suspicious for, but not yet diagnostic, of lymphoma and repeat biopsy was recommended.

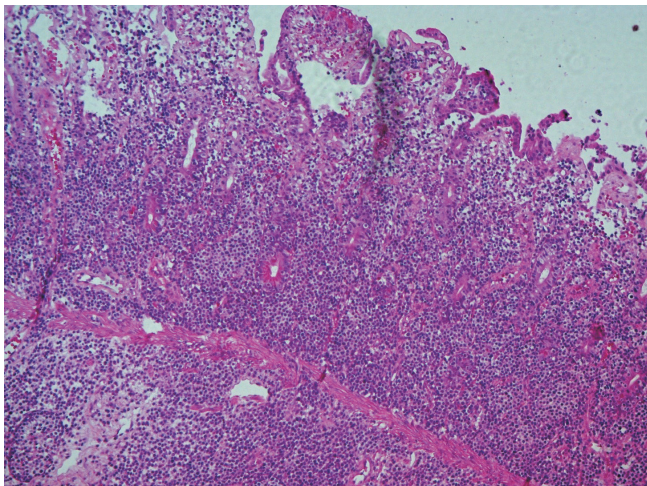


Fig. 3. Pathohistological tissue sample from ileocaecum resection material Haematoxylin eosin, 50x. Mucosal and submucosal infiltration with monotonous medium and small-sized lymphocytes. Increased numbers of intraepithelial lymphocytes.

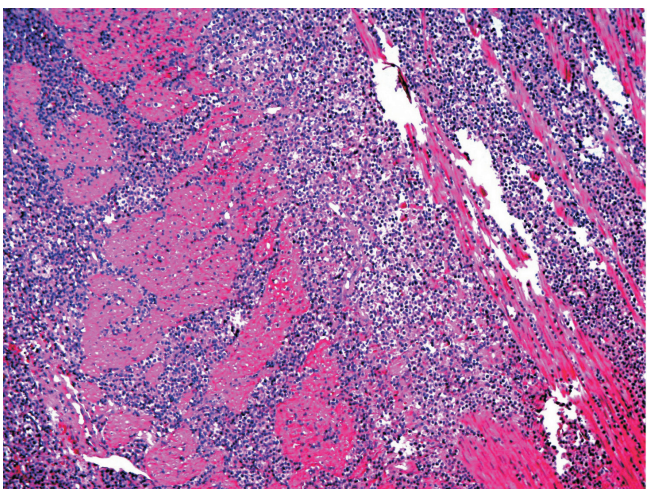


Fig. 4. Pathohistological tissue sample from ileocaecum resection material EnVision, Haematoxylin eosin, 50x. Deep monotonous infiltration with medium- and small-sized lymphocytes in muscle layer.

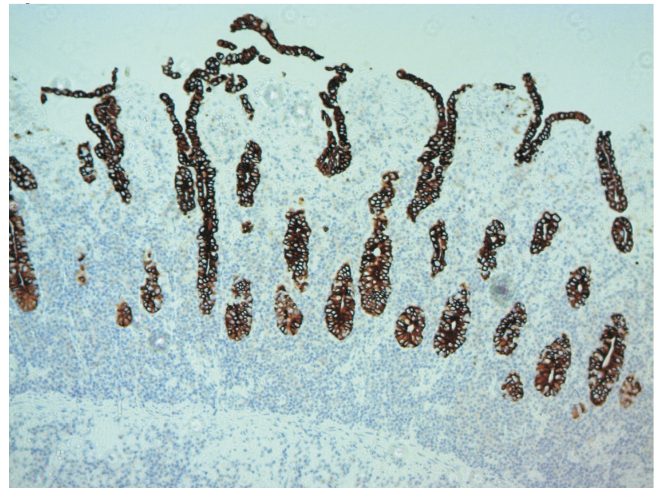


Fig. 5. Pathohistological tissue sample from ileocaecum resection material CK AE1/AE3+, EnVision, 50x. Small intestines mucosa with marked villous atrophy.



Fig. 6. Pathohistological tissue sample from ileocaecum resection material CD 3, EnVision, 100x. Lymphocyte infiltrate shows intensive staining for T-cells marker — CD3(+).

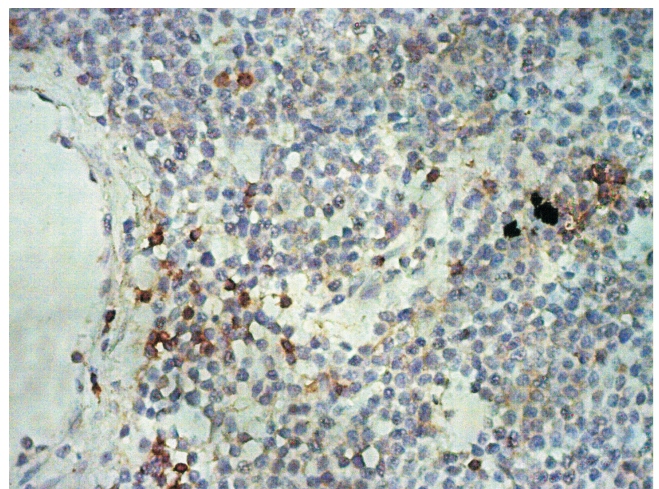


Fig. 7. Pathohistological tissue sample from ileocaecum resection material. CD 4, EnVision, 400x. T-lymphocytes are CD4 negative. Expression detected only in % cells

The patient underwent an ileostomy procedure. The stoma was fully functioning. Surgical drain was evacuated on day 7 of the postoperative period. On day 10, the patient experienced sudden dyspnoea and was transferred to the ICU, where she died. Based on clinical, laboratory, endoscopic and histological data, the following final diagnosis was established: highly malignant PTCL-NOS with small intestine involvement; microperforation of the small intestine; diffuse serofibrinous peritonitis.

DISCUSSION

Cases of peripheral T-cell lymphomas initially misdiagnosed or easily confused with inflammatory bowel disease (IBD) have been reported previously (Tsai *et al.*, 2013; Sun *et al.*, 2014; Tian *et al.*, 2019, Wan Ahmad Kammal *et al.*, 2019). With GI tract involvement, patients present with nonspecific symptoms that usually require endoscopic and radiological studies for correct diagnosis. In our case, CD was mimicking lymphoma clinically, endoscopically and histologically. The final diagnosis of PTCL-NOS was established only post-mortem. Therefore, when CD is suspected, physicians shouldn't forget about the differentiating it from lymphoma.

PCTLs involving the GI have been studied radiologically. Zhu *et al.* (2012) reported 15 patients with GI involvement, most commonly in the colon ($n = 5$) and stomach ($n = 6$). As in our case, bowel wall thickening was the predominant finding on CT scan in all cases, except one intestinal PTCL that presented as a polypoid mass. Mendelson and Fermoyle (2006) reported bowel wall thickening in PTCL, which has a wide differential diagnosis including CD, and a nodular pattern with submucosal nodules with or without ulceration, requiring differentiation from CD and multiple metastases (Mendelson and Fermoyle, 2006). In a study by Byun *et al.*, PTCL involved the stomach in three patients, small intestine in eight, both the stomach and small intestine in one, and the sigmoid colon in two patients; multifocal involvement was seen in three patients. The authors recommend including PTCL in the list of differential diagnoses based on a CT scan when the duodenum or the jejunum is a preferential site of intestinal involvement, when multifocal bowel involvement is seen, and there is evidence of bowel perforation or diffuse peritonitis (Buyn *et al.*, 2003). Magnetic resonance enterography may be required to clarify the diagnosis in cases of small intestinal lymphoma (Matysiak-Budnika *et al.*, 2017).

Endoscopically, ileocolonic malignant lymphomas classify into fungating, ulcerative, infiltrative, ulcerofungating, and ulceroinfiltrative types (Myung *et al.*, 2003). In colorectal primary T-cell lymphomas, ulcerative lesions are the most common (Yu *et al.*, 2014). Lee *et al.* (2001) reported six cases of PTCL of the colon, when four patients were initially diagnosed with either IBD or tuberculous colitis based on endoscopic features, and advised to suspect T-cell lymphoma in a patient who had the radiologic features of IBD with extensive ulcerations that was refractory to treatment

(Lee *et al.*, 2001). Results suggest the need for additional studies to determine specific endoscopic features that point towards a diagnosis of primary GI T-cell lymphoma (Kim *et al.*, 2014). Repeated colonoscopic biopsy at multiple sites and immunohistochemical staining are warranted to make a definitive diagnosis (Wan Ahmad Kammal *et al.*, 2019). In case of small intestine, lymphoma enteroscopy (if biopsies are mandatory to establish the diagnosis) or capsule endoscopy should be performed (Matysiak-Budnika *et al.*, 2017).

Diagnostic difficulties present themselves not only in endoscopy, but also in histological analysis, as it is sometimes difficult to distinguish between lymphoma and dense inflammatory infiltration. There is a need for a high index of suspicion to differentiate lymphoma from inflammation. As for immunohistochemistry, PTCL-NOS shows frequent loss of one or more of the pan T-cell antigens (CD2, CD3, CD5, CD7). The most common is CD4+ (65% of cases), CD8+ is present in 15% of cases. PTCL can also express CD30 and cytotoxic markers, including TIA1, granzyme B, perforin, CD56 and CD5 (Rodriguez-Abreu *et al.*, 2008). There are reports indicating that cytotoxic marker expression may be associated with a poor outcome (Iqbal *et al.*, 2010). Epstein-Barr virus is found in approximately 30% of all cases of PTCL-NOS and may be associated with a more aggressive course (Dupuis *et al.*, 2006). PTCL-NOS usually lacks the follicular T-helper phenotype (CD10+, Bcl6+, PD1+, CXCL13+) with the exception of the follicular variant. Proliferation is usually high and Ki-67 rates exceeding 80% are also associated with a worse outcome (Went *et al.*, 2006). In our case, immunohistochemical stains were positive for CD3, CD5, BCL2, LCA, Ki67 (50–70 %), focally positive for CD4, CD8 and negative for CyclinD1, CD79a, CD20, CD30.

One of the differential diagnoses for PTCL-NOS among T-cell lymphomas is EATL (previously termed enteropathy-associated T-cell lymphoma, type I) — a rare, coeliac-disease associated tumour. EATL predominantly affects the small bowel, but can sometimes present in the stomach and colon. What was previously considered EATL type II, without pre-existing coeliac disease, is now defined as a separate entity among aggressive GI PTCLs — monomorphic epitheliotropic intestinal T cell lymphoma (MEITL) (Matutes, 2018). The two entities — PTCL-NOS and EATL — are indistinguishable clinically, and immunochemistry is not always helpful. EATL tumour cells are CD3+, CD5-, CD8+/-, CD4-, CD56-, CD103+ and contain cytotoxic granule associated proteins (Swerdlow *et al.*, 2017). MEITL display CD8, CD56 and SYK expression that is generally not seen in EATL (Mutzbauer *et al.*, 2018). Morphologically, EATL consists of irregular intermediate to large cells, necrosis and prominent background inflammatory infiltrate. In our case, the patient did not have a diagnosed coeliac disease, atypical cells were regular and small to medium in size, and expressed CD4 and CD5 antigens, which is not characteristic of EATL.

Additionally, possible new diagnostic and disease severity markers are being investigated. Shen *et al.* (2019) report

elevated levels of plasma programmed death 1 protein in patients with PTCL, where higher levels seem to be associated with clinical aggressiveness of the disease (Shen *et al.*, 2019). NKp46 is an emerging diagnostic marker of GI T-cell lymphomas and a potential therapeutic target. Data from the CELAC study show it is only rarely expressed in normal lymphoid tissues, whereas expression was detected in 83% of EATL (n = 24/29) and in 100% of MEITL (n = 4/4). NKp46 was rarely expressed in PTCL-NOS (n = 1/10) (Cheminant *et al.*, 2019).

As in other cases described in the literature, we also had diagnostic difficulties with clinical condition, endoscopy and histological analysis, because all findings were suggesting CD, which is a more common diagnosis. Performing a capsule endoscopy or enteroscopy might have led to the correct diagnosis earlier (Hashimoto and Matsuda, 2019). Immunohistochemical findings pointed to intestinal lymphoma, but repeated biopsies were not possible due the deterioration of the patient's condition. Because of diagnostic difficulties and common extranodal presentation of PTCL, the correct diagnosis is often significantly delayed. Lee *et al.* (2001) reported delay in diagnosis from eight months to four years from the initial presentation of the disease. The correct diagnosis was finally made after surgical resection of the bowel in three patients and repeated biopsies in one (Lee *et al.*, 2001).

CONCLUSION

Differentiation of T-cell lymphoma from CD continues to be challenging. Incomplete response to CD treatment, aggressive disease course with bowel perforation and multifocal GI involvement might be reasons to review IBD diagnosis. Small bowel investigation using capsule endoscopy and/or enteroscopy, as well as repeatedly performed biopsies at multiple GI sites, could help to rule out a haematological malignancy. Immunohistochemistry is essential to distinguish between GI lymphoma types and provide the most appropriate treatment. Due to multiple diagnostic difficulties, collaboration between all medical care units is crucial.

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T ŠŪNU LIMFOMAS UN KRONA SLIMĪBAS SLIMĪBAS DIFERENCIĀLDIAGNOSTIKA: GADĪJUMA ZIŅOJUMS

Jau daudzus gadus pastāv bažas, ka iekaisīgai zarnu slimībai ir paaugstināts limfomas attīstības risks. Tajā pašā laikā kuņģa zarnu trakta limfoma dažkārt tiek kļūdaini diagnosticēta kā Krona slimība. Šajā klīniskajā gadījumā tiek atspoguļots intestinālas T-šūnu limfomas gadījums, kas sākotnēji diagnosticēts kā Krona slimība, tiek akcentēti diagnostiskie izaicinājumi, ko rada ekstranodālas limfomas. 68 gadus vecas sievietes sūdzības (caureja, sāpes vēderā, slikta apetīte un ievērojams svara zudums), kolonoskopijas dati un sākotnējā histoloģiskā aina atbilda iekaisīgai zarnu slimībai — tika noteikta Krona slimības diagnoze, tika uzsākta ārstēšana ar sulfasalazīnu, kas deva klīniskās simptomātikas uzlabošanu. Astoņus mēnešus pēc sākotnējās diagnozes noteikšanas pacientei parādījās pēkšņas sāpes vēderā. Laparotomijas laikā tika atklātas nekrozes zonas tievajā un resnajā zarnā, un tika izveidota ileostoma. KomPLICĒTA PĒCOPERĀCIJAS PERIODA 10. dienā iestājās *exitus letalis*. Tievās un resnās zarnas pēcnāves histopatoloģiskā izmeklēšana atklāja augstas malignitātes limfomu (PTCL-NOS). Intestinālas T-šūnu limfomas un Krona slimības diferenciāldiagnostika aizvien ir izaicinājums, jo klīniskā, endoskopiskā, radioloģiskā un histopatoloģiskā aina var būt līdzīga abu saslimšanu gadījumā. Lai savlaicīgi diagnosticētu intestinālu limfomu, svarīga ir rūpīga multidisciplināra pieeja pacientam un zināšanas par šo reto patoloģiju.