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Editorial Board of *World Journal of Gastroenterology*, Haruhiko Sugimura, MD, PhD, Director, Sasaki Institute, Sasaki Foundation, Tokyo 101-0062, Japan. hsugimur@po.kyoundo.jp

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CASE REPORT

Drug-induced entero-colitis due to interleukin-17 inhibitor use; capsule endoscopic findings and pathological characteristics: A case report

Keita Saito, Kiichiro Yoza, Shinichiro Takeda, Yoshihiro Shimoyama, Ken Takeuchi

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Keita Saito, Kiichiro Yoza, Shinichiro Takeda, Yoshihiro Shimoyama, Ken Takeuchi, Department of Gastroenterology, Tsujinaka Hospital Kashiwano-Ha, Kashiwa 277-0871, Japan

Corresponding author: Keita Saito, MD, Doctor, Department of Gastroenterology, Tsujinaka Hospital Kashiwano-Ha, No. 178-2 Wakashiba, Kashiwa 277-0871, Japan.
keisaitou.ymgt@gmail.com

Abstract

BACKGROUND

Interleukin-17 (IL-17) inhibitors are known to cause exacerbation or new onset of inflammatory bowel disease upon administration. However, few reports have described characteristic endoscopic and histopathologic findings, and no small intestinal lesions have been reported so far.

CASE SUMMARY

A woman in her 60s with psoriasis was administered ixekizumab (IXE), an anti-IL-17A antibody, for the treatment of psoriasis. Twenty months after commencing treatment, the patient visited our hospital because of persistent diarrhea. Blood tests performed at the time of the visit revealed severe inflammation, and colonoscopy revealed multiple round ulcers throughout the colon. A tissue biopsy of the ulcer revealed infiltration of inflammatory cells and granuloma-like findings in the submucosal layer. Capsule endoscopy revealed multiple jejunal erosions. After the withdrawal of IXE, the symptoms gradually improved, and ulcer reduction and scarring of the colon were endoscopically confirmed.

CONCLUSION

To the best of our knowledge, 17 reports have documented IL-17 inhibitor-induced entero-colitis with endoscopic images, endoscopic findings, and pathological characteristics, including the present case. Nine of these cases showed diffuse loss of vascular pattern, coarse mucosa/ulcer formation in the left colon, and endoscopic findings similar to those of ulcerative colitis. In the remaining eight cases, discontinuous erosions and ulcerations from the terminal ileum to the rectum were seen, with endoscopic findings similar to those of Crohn's disease. In this case, the findings were confirmed by capsule endoscopy, which has not been previously reported.

Key Words: Interleukin-17 inhibitor; Ixekizumab; Drug-induced entero-colitis; Capsule endoscopy; Case report

Core Tip: While Interleukin-17 (IL-17) inhibitors are effectively used in the treatment of psoriasis, psoriatic arthritis, and ankylosing spondylitis, they are ineffective in patients with Crohn's disease (CD) and can worsen their condition. To the best of our knowledge, we present capsule endoscopic images of IL-17 inhibitor-induced entero-colitis for the first time, suggesting that IL-17-induced inflammatory lesions may be distributed in the proximal small bowel, unlike CD lesions. We also compared the endoscopic and pathological features of IL-17 inhibitor-induced entero-colitis with those previously reported.

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INTRODUCTION

Interleukin-17 (IL-17) inhibitors, such as ixekizumab (IXE) and secukinumab, are a class of molecular-targeted therapies used to treat psoriasis, psoriatic arthritis, and ankylosing spondylitis. IL-17 is a type of inflammatory cytokine produced by helper T cells and is known not only to induce local inflammation in the human body but also to be involved in host infection defense against pathogens in the skin and intestinal epithelium[1]. In patients with both psoriasis and Crohn's disease (CD), biopsy specimens of lesions express high levels of IL-17[2,3]. Therefore, IL-17 inhibitors were hypothesized to be effective in treating psoriasis and CD. However, IL-17 inhibitors are only effective in psoriasis; in patients with CD, IL-17 inhibitors are ineffective and exacerbate the disease[4]. Furthermore, in clinical trials of IL-17 inhibitors in inflammatory bowel disease (IBD), rheumatic diseases, and dermatological diseases, exacerbations or new-onset IBD have been reported at a frequency of 0.4%[5]. The mechanism underlying this seemingly contradictory adverse reaction remains unclear.

CASE PRESENTATION

Chief complaints

A woman in her 60s with diarrhea and anorexia.

History of present illness

Gastrointestinal symptoms appeared 24 mo after IXE was started for the treatment of psoriasis.

History of past illness

The patient was diagnosed with psoriatic arthritis by her family physician and started on IXE. However, anorexia and diarrhea appeared 20 mo after treatment initiation. After conservative treatment by her family doctor, her symptoms did not improve, and she visited our hospital 24 mo after IXE initiation for a close examination and treatment.

Personal and family history

Her medical history included type 1 diabetes at the age of 35 and hypothyroidism at the age of 50 years, each of which was medically managed by her family physician. No family history of IBD was reported; her father had gastric cancer, and her mother had diabetes. The injectable medications used were insulin and IXE for diabetes and psoriasis, respectively.

Physical examination

The patient was conscious but noticeably emaciated, appeared weakened, and walked with a limp. She had a body temperature of 36.0 ℃ and 114/52 mmHg of blood pressure. The skin of the upper extremities was fragile, with epidermal exfoliation of the right forearm. Multiple scars were observed on the upper arm and mild deformities and swelling of the hand joints.

Laboratory examinations

On admission, blood biochemistry tests showed anemia with a hemoglobin level of 10.4 g/dL and hypoalbuminemia with an albumin level of 2.8 g/dL. She was also dehydrated, with a blood urea nitrogen level of 28.2 mg/dL and

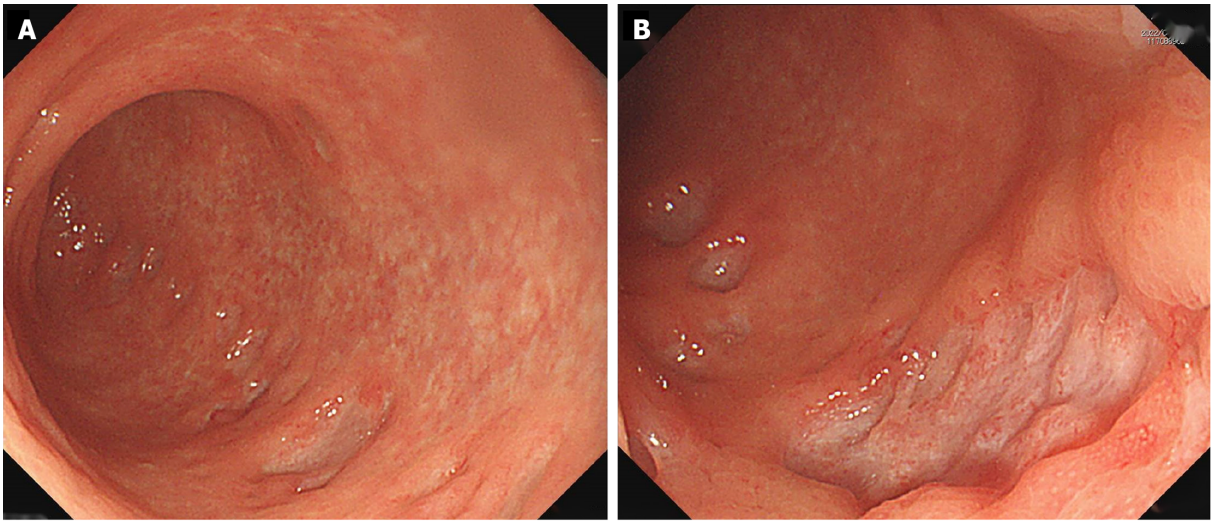

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Figure 1 Colonoscopy findings at admission. A: Distant view of colon; B: Close-up of ulcer. Multiple round punctate ulcers with a longitudinal trend from the cecum to the rectum are observed. The intervening mucosa of the ulcers is preserved, and the ulcers do not coincide with the mesenteric attachment side.

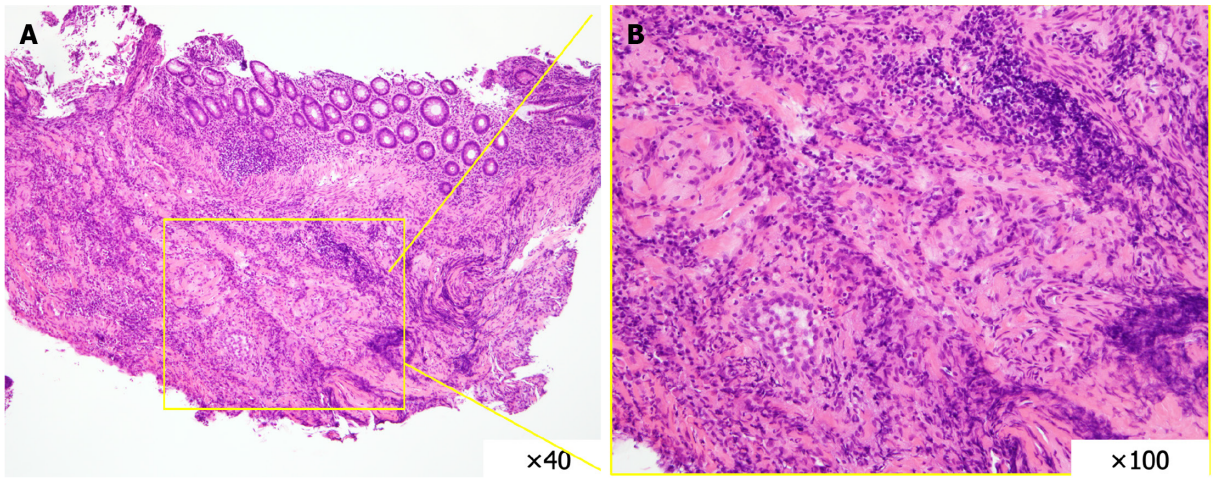

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Figure 2 Pathological findings at admission. A: Hematoxylin-eosin (HE) stained specimen at 40 × magnification; B: HE stained specimen at 100 × magnification. The mucosa is erosive and infiltrated with inflammatory cells, predominantly lymphocytes. The submucosa shows granulomatous collagen fibers and fibroblast proliferation.

creatinine of 0.8 mg/dL and had high inflammation with a C-reactive protein level of 15.3 mg/dL. Leucine-rich alpha-2 glycoprotein level was 44.1 μg/mL and fecal calprotectin level was also high at 7357 mg/kg, suggesting strong intestinal inflammation.

Imaging examinations

Computed tomography revealed edematous wall thickening of the intestinal tract, continuous from the ascending colon to the rectum.

Further diagnosis workup

Colonoscopy revealed multiple round, punched-out ulcers with a longitudinal trend from the cecum to the rectum (Figure 1). The intervening mucosa of the ulcer was nearly normal, and the ulcer did not coincide with the mesenteric attachment. A biopsy of the ulcer showed mucosal erosion, lymphocyte-dominated inflammatory cell infiltration, and regenerative epithelial growth (Figure 2). Submucosal fibroblast and collagen fiber proliferation were also present-a granuloma-like finding similar to that of CD. An upper gastrointestinal endoscopy revealed reflux esophagitis and chronic gastritis. The gastric mucosa exhibited scattered erythema and erosions; however, no specific abnormalities were observed in the duodenum. Capsule endoscopy revealed multiple jejunal erosions (Figure 3). The erosions were scattered on the proximal side of the jejunum; each erosion was shallow and < 1 cm in size, and hematin adhesions were visible on the surface. However, stool culture and *Clostridioides difficile* toxin tests were negative, as were cytomegalovirus antigen

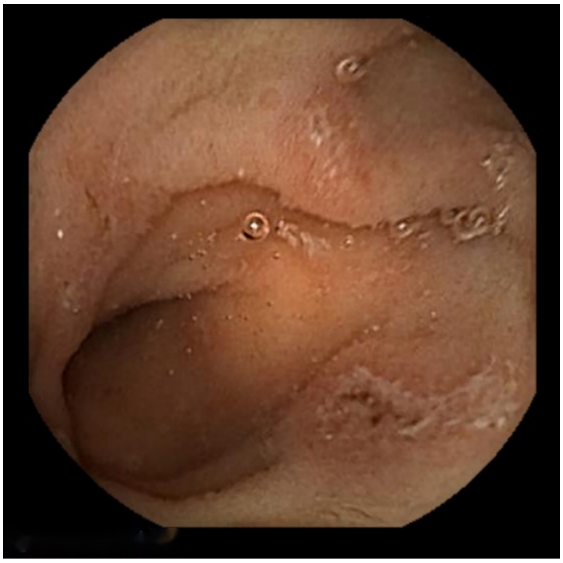

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Figure 3 Capsule endoscopy findings. Scattered erosions are seen in the jejunum. The erosions were scattered on the proximal side of the jejunum; each erosion was shallow and < 1 cm in size, and hematin adhesions were visible on the surface.

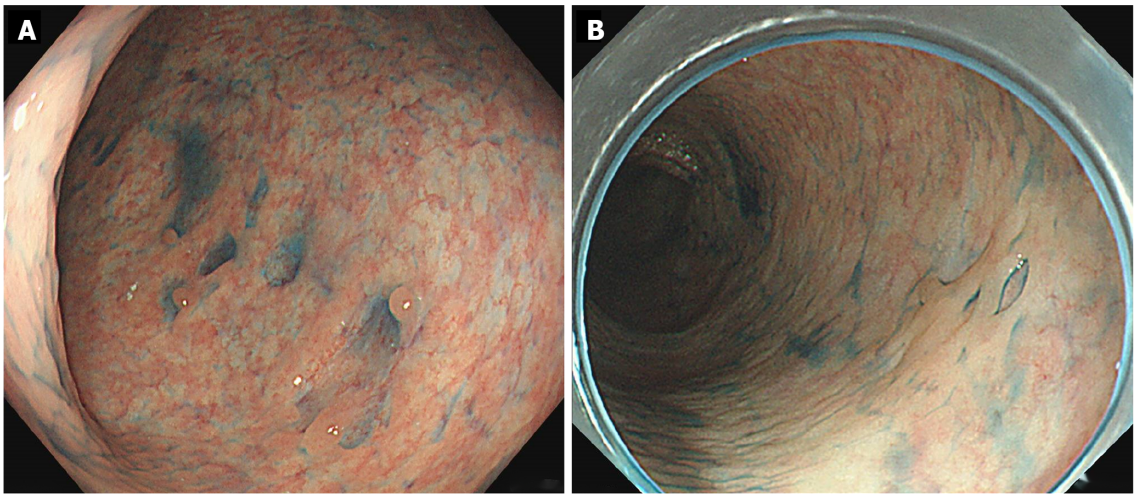

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Figure 4 Colonoscopy findings after drug withdrawal. A: At three weeks, there is shrinkage of ulcers; B: At four months, all ulcers have disappeared and scarring is observed.

and polymerase chain reaction tests and interferon-gamma release assays.

FINAL DIAGNOSIS

As no episodes of irradiation or introduction of other new drugs occurred, we suspected drug-induced due to IXE.

TREATMENT

First, we monitored the patients' progress during drug withdrawal and intestinal rest after fasting. Multiple erosions in the upper jejunum were also observed; therefore, the patient commenced on bonoprazan fumarate, a potassium-competitive acid blocker. Due to the lack of improvement in symptoms, steroid administration was considered, and a gradual improvement in abdominal symptoms was observed. Three weeks after withdrawal, endoscopy revealed shrinkage of the ulcer and scarring (Figure 4A). Although her abdominal symptoms resolved, her skin and joint symptoms worsened, and she was started on Risankizumab by her family physician for psoriasis treatment.

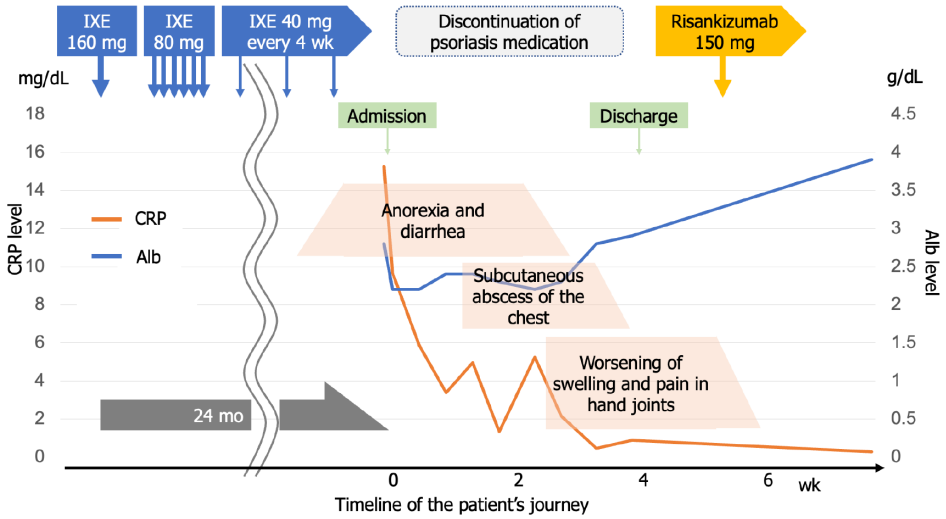

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Figure 5 Albumin (Alb) and C-reactive protein (CRP) levels plotted against the timeline of the patients' journey. The patient visited our hospital 24 mo after the start of the ixekizumab (IXE) administration. In the graph, CRP is shown on the left vertical axis and Alb on the right vertical axis. Abdominal symptoms gradually improved with IXE withdrawal, but skin and joint symptoms tended to worsen. After 4 wk of hospitalization, the patient was discharged home, as the endoscopy showed that the ulcer had healed and the patient was able to eat adequately. After discharge, risankizumab was introduced to control skin and joint symptoms, and the patients' condition stabilized. IXE: Ixekizumab; CRP: C-reactive protein; Alb: Albumin.

OUTCOME AND FOLLOW-UP

The abdominal, skin, and joint symptoms remained stable, and endoscopy performed 4 mo after IXE withdrawal confirmed the disappearance of all ulcers and scarring (Figure 4B). As abdominal symptoms improved, capsule endoscopy was not performed again due to a lack of patient consent, and bonoprazan fumaric acid was also discontinued. The clinical course from IXE initiation to the present is illustrated in Figure 5.

DISCUSSION

Drug-induced entero-colitis caused by IL-17 inhibitors is well known in the field of dermatology; however, very few reports have described all the endoscopic images, endoscopic features, and pathological characteristics of this condition. To the best of our knowledge, only 16 cases have been reported thus far[5-20]; we reviewed 17 cases, including our own (Table 1). Nine reported cases showed ulcerative colitis (UC)-like findings characterized by circumferential loss of vascular pattern and coarse mucosa/ulceration in the left colon, and eight reported cases with CD-like findings characterized by discontinuous erosion/ulceration from the terminal ileum to the rectum. All patients who presented with CD-like endoscopic findings after IXE administration had granulomas. In almost all the reports, the disease prognosis appeared to be good, with improvement in abdominal symptoms after the administration of steroids or molecularly targeted drugs. Only one patient with UC-like endoscopic findings after IXE administration required surgery because of a lack of improvement with drug administration.

Another case similar to CD with multiple ulcers of a similar round shape, as in the present case, has also been reported. However, in the present case, the ulcers tended to be longitudinally arranged and did not coincide with the mesenteric attachment side, which is atypical of CD. Furthermore, no reports have indicated improvement in abdominal symptoms with drug discontinuation alone, as in this case. In the present case, the various test results allowed us to promptly identify IXE as the suspected drug, and we surmised that excessive therapeutic intervention could be avoided. It should be noted that the introduction of a new drug may be necessary to manage the primary disease after drug withdrawal, and close communication with the dermatologist is important.

The association between psoriasis and IBD should be investigated in future studies. It has been reported that 1%-2% of patients with psoriasis have IBD[21]. Coincidentally-timed events during the initiation or administration of IL-17 inhibitors highlighted that IBD cannot be excluded.

CONCLUSION

At the very least, we should always check for IBD-related symptoms and family history before administering IL-17 inhibitors and suggest a screening colonoscopy if possible. Here, we report, for the first time, the capsule endoscopic findings of IL-17 inhibitor-induced entero-colitis. We also compared the endoscopic and pathological features of IL-17 inhibitor-induced entero-colitis with those previously reported. We believe that these findings will be useful for dermato-

Table 1 Patient background and endoscopic and pathological findings of entero-colitis after interleukin-17 administration

Year	Ref.	Age	Sex	Primary disease	Drug	Time to onset	IBD	Endoscopic findings	Pathological findings	Treatment and course
2017	Shiga *et al* [5]	56	M	Psoriasis	SEC	8 wk	CD	Longitudinal ulcer of the ileum and round ulcer of the esophagus	Nonspecific inflammatory cell infiltration	Improved with prednisolone 40 mg/d
2018	Philipose *et al*[6]	31	M	Psoriasis	IXE	3 mo	UC	Loss of vascular permeability throughout the sigmoid colon, erythematous coarse mucosa, ulcer	Lymphoplasmacytic infiltration	Mesalamine and methylprednisolone did not improve, but IFX administration improved
2018	Wang *et al* [7]	41	F	Psoriasis	SEC	1 wk	UC	Coarse mucosa and deep-burrowing ulceration of the entire sigmoid colon	Cryptitis, erosions, lymhoplasmacytic infiltration	Improved with methylprednisolone 40 mg/d and cyclosporine 2 mg/kg
2018	Ehrlich *et al*[8]	42	M	Ankylosing spondylitis	SEC	6 wk	UC	Deep ulcers and fragile mucosa of the transverse and sigmoid colon	Cryptitis, crypt abscess, loss of crypts	No improvement with solumedrol, improved after introduction of IFX
2019	Smith *et al* [9]	42	M	Psoriasis	IXE	12 wk	CD	Deep rounded punctate ulcers of the transverse and descending colon	Pancolitis with rare granuloma	No improvement with solumedrol, improved after introduction of IFX
2019	Uchida *et al*[10]	41	F	Psoriasis	SEC	4 mo	UC	Easy bleeding edematous mucosa of rectum to sigmoid colon, erosions, ulcers	High degree of inflammatory cell infiltration into the stroma and crypt abscess	Improved with mesalazine 2400 mg/d
2019	Achufusi *et al*[11]	39	M	Psoriasis	SEC	6 mo	UC	Ulceration of the splenic flexure, moderate to severe active colitis, ulceration at 30 cm, and active colitis in the rectum	Atrophy of the crypts, decreased goblet cells, cryptitis, crypt abscess	No improvement with steroids, improved after introduction of IFX
2019	Johnston and Veettil [12]	27	M	Ankylosing spondylitis	SEC	4 mo	UC	Multiple ulcers and moderate inflammation, sigmoid colon	Crypt abscess	No improvement with mesalazine and hydrocortisone, improvement with introduction of IFX
2019	Haidari *et al*[13]	69	M	Psoriatic arthritis	SEC	18 mo	CD	Multiple ulcers of the terminal ileum	Neutrophil infiltration of the epithelium of the crypts, no granuloma	Originally asymptomatic
2020	Nazarian *et al*[14]	48	F	Psoriasis	IXE	12 wk	CD	Mild erythema and punctate ulcerations in the terminal ileum	Active inflammation with the presence of granuloma	Improved with budesonide administration
2020	Varga *et al* [15]	52	M	Psoriasis	SEC	2 wk	UC	Loss of vascular permeability of sigmoid colon, ulcer	Lymphocytic infiltration of lamina propria, cryptitis, crypt abscess	Improved with prednisone 60 mg/d and mesalazine 3200 mg
2020	Gallego *et al*[16]	42	M	Psoriasis	IXE	2 wk	CD	Aphthous erosions and patchy ulcers of the rectum to cecum and terminal ileum	Cryptitis, crypt abscess, non-caseating granuloma	Improved with systemic corticosteroid administration
2021	Ali *et al*[20]	70	F	Psoriasis	SEC	1 mo	UC	Ulcerated and edematous mucosa in sigmoid colon	Acutely and chronically inflamed granulation tissue with extensive plasma cell infiltrate	Intravenous methylprednisolone
2022	Kakizoe *et al*[17]	65	M	Psoriasis	SEC	15 mo	CD	Deep ulcers of the cecum and transverse colon	No description	Hematochezia persisted after drug discontinuation and improved after induction of ADA
2022	Morosanu *et al*[19]	42	F	Psoriasis	IXE	1 wk	UC	Continuous congestive, friable rectal and colonic mucosa, spontan-	Neutrophilic inflammatory infiltrate disposed irregularly, edema and congestion, decrease of the crypts mucose-	Total colectomy with ileostoma and rectum preservation

								eously bleeding, deep and large ulcerations	cretion and crypt's abscesses	
2023	Khouri *et al*[18]	38	F	Psoriatic arthritis	SEC	1 mo	CD	Small ulcerations throughout the entire lumen of the terminal ileum and the cecum	Minimal architecture distortion in the large bowel mucosa, along with focal acute colitis	Initiated with prednisone and SEC was switched to IFX
2022	Our case	69	F	Psoriatic arthritis	IXE	21 mo	CD	Multiple round punctate ulcers throughout the colon. Capsule endoscopy shows multiple erosions in the jejunum	Inflammatory cell infiltrate, predominantly lymphocytes. Granulomatous fibroblasts and collagen fibers in the submucosa	Improvement only with drug discontinuation and fasting bowel rest

CD: Crohn's disease; SEC: Secukinumab; M: Male; F: Female; IBD: Inflammatory bowel disease; IXE: Ixekizumab; UC: Ulcerative colitis; ADA: Adalimumab.

logists and gastroenterologists in clinical practice.

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FOOTNOTES

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Country/Territory of origin: Japan

ORCID number: Keita Saito 0000-0003-2740-4518; Shinichiro Takeda 0000-0002-6302-5073.

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