Dermatology Practical & Conceptual

Retrospective Analysis of Demographic and Clinical Data of Psoriasis Patients Who Developed Inflammatory Bowel Disease Under Secukinumab and Ixekizumab Treatment

Uğur Kılıçaslan[1], Fatma Aslı Hapa[1]

1 Department of Dermatology, Faculty of Medicine, İzmir Democracy University, İzmir, Turkey

Key words: IL-17A inhibitors; Inflammatory bowel disease; Ixekizumab; Psoriasis; Secukinumab

Citation: Kılıçaslan U, Hapa FA. Retrospective Analysis of Demographic and Clinical Data of Psoriasis Patients Who Developed Inflammatory Bowel Disease Under Secukinumab and Ixekizumab Treatment. *Dermatol Pract Concept*. 2026;16(2):7125. DOI: https://doi.org/10.5826/dpc.1602a7125

Accepted: March 18, 2026; **Published:** April 2026

Funding: None.

Competing Interests: None.

Authorship: All authors have contributed significantly to this publication.

Corresponding Author: Uğur Kılıçaslan, MD, İ Department of Dermatology, Faculty of Medicine, İzmir Democracy University, İzmir, 35390, Turkey. ORCID: 0009-0000-4398-4431. E-mail: kilicaslanugur@gmail.com

ABSTRACT

Introduction: The aim of this study was to retrospectively investigate the frequency of inflammatory bowel disease (IBD) and the demographic and clinical characteristics of psoriasis (PsO) patients exposed to IL-17A inhibitors.

Method: Health data of 2,264 PsO patients who had received at least one dose of ixekizumab or secukinumab were retrospectively reviewed. Demographic data, clinical characteristics, symptoms consistent with IBD and confirmed IBD diagnoses of the patients were collected. Among the patients presenting with IBD-related symptoms during follow-up, those diagnosed with IBD after gastroenterology consultation were additionally recorded.

Results: Among the 2,264 patients, 28 individuals (1.2%) reported gastrointestinal symptoms consistent with IBD (diarrhea, abdominal pain, bloody stool). The median time from initiation of IL-17A inhibitor therapy to symptom onset was 5.8 months. Of the symptomatic patients, 18 were female (64.3%) and 10 were male (35.7%). Fifteen patients (53.6%) were secukinumab users, and 13 (46.4%) were ixekizumab users; there was no significant difference between the drug groups in terms of symptom frequency (P=0.67). Following gastroenterological evaluation, seven patients (0.3%) were diagnosed with IBD, four of whom were secukinumab and three were ixekizumab users (P=0.72). Among the confirmed cases, three had ulcerative colitis and four had Crohn's disease.

Conclusion: Gastrointestinal symptoms were observed in 1.2% of the study population, while the rate of confirmed IBD after gastroenterological evaluation was 0.3%. This rate is similar to that of the general population. The distribution of ulcerative colitis and Crohn's disease cases was equal between ixekizumab and secukinumab users. Monitoring for gastrointestinal symptoms during the first six months of treatment is important.

Introduction

Interleukin (IL)-17A is a proinflammatory cytokine that plays a crucial role in the pathogenesis of autoimmune diseases and the maintenance of the intestinal mucosal barrier [1]. Secukinumab and ixekizumab, which target this cytokine, are widely and effectively utilized in the treatment of moderate-to-severe psoriasis (PsO) [2].

Physiologically, IL-17A is secreted in the lamina propria of the intestinal wall and plays a significant role in maintaining intestinal mucosal integrity and immunity against pathogens [3]. Furthermore, elevated levels of IL-17A in patients with inflammatory bowel disease (IBD) have led to the hypothesis that IL-17A inhibitors could serve as a therapeutic target in IBD. However, cases of new-onset IBD or exacerbations of existing IBD have been reported in patients treated with IL-17A inhibitors [4-10]. Consequently, the potential of IL-17A inhibitors to induce colitis remains controversial and has not been fully elucidated [11,12].

In clinical practice, these cases typically present with symptoms such as diarrhea, bloody stools, abdominal pain, and fever. Additionally, they are accompanied by leukocytosis, elevated C-reactive protein (CRP), and increased fecal calprotectin levels [13]. Moreover a strong association is thought to exist between PsO and IBD [14-17]. Meta-analyses have found that individuals diagnosed with PsO carry a significantly higher risk for developing both Crohn's disease (CD) and ulcerative colitis (UC) [18].

Objectives

In light of this information, this study aimed to determine the frequency of gastrointestinal symptoms compatible with IBD and diagnosed IBD cases in PsO patients using the IL-17A inhibitors secukinumab or ixekizumab, to evaluate the demographic and clinical characteristics of these cases, and to investigate the differences between treatment groups.

Materials and Methods

The study was conducted with a retrospective, single-center design. Health records of a total of 2,264 patients diagnosed with PsO, who were treated at the İzmir Democracy University, Buca Seyfi Demirsoy Training and Research Hospital, Dermatology Clinic between 2020 and 2025 and received at least one dose of secukinumab or ixekizumab biologic agent therapy were screened retrospectively. Ethics committee approval was obtained prior to the study (decision no: 2025/503, date: 24 September 2025).

Demographic data (age, sex, etc.) and clinical data (type of biologic agent, symptoms, etc.) of the patients were recorded from file archives and computer systems. None of the patients included in the study received any other biologic agent or systemic immunosuppressive therapy (TNF inhibitors, IL-12/23 inhibitors, methotrexate, cyclosporine, apremilast, etc.) during the period examined. This was ensured to eliminate the effects of factors other than IL-17A inhibitors on the results. Additionally, during the baseline evaluation, gastrointestinal history was thoroughly investigated, and patients with known inflammatory bowel disease, a significant family history, or clinically significant gastrointestinal pathology (e.g., diverticular disease) were not included in IL-17A inhibitor therapy.

Symptom Screening

During the study period, patient files were screened for symptoms compatible with IBD, such as diarrhea, abdominal pain, and bloody stools. Patients exhibiting these symptoms were identified, and the number of cases showing gastrointestinal complaints was determined. Symptoms documented in clinical notes, admission records, or patient complaints within the study period were included in the screening: diarrhea (≥3 watery stools per day for ≥3 days), abdominal pain (clinically significant, recorded by a physician), bloody stools (hematochezia/rectal bleeding), weight loss, and fever.

Diagnostic Confirmation

A gastroenterology consultation was requested for every patient in whom gastrointestinal symptoms were detected. A definitive IBD diagnosis (Ulcerative Colitis – UC and Crohn's Disease - CD) was considered valid for cases diagnosed by a gastroenterology specialist. The diagnosis type was recorded as UC or CD. In this process, clinical and sociodemographic data of only those cases diagnosed with IBD following the gastroenterological evaluation were recorded separately.

Statistical Analysis

Data were analyzed using SPSS software. Continuous variables are presented as mean ± standard deviation or median [IQR], and categorical variables are presented as numbers and percentages. The difference in IBD incidence rates between different treatment groups was evaluated using the chi-square test ($P<0.05$), and the results were compared with the general IBD incidence rates reported in the international literature. Due to the retrospective design of the study, person-time calculations were not performed to calculate incidence rates or relative risks; only percentage differences between groups were interpreted.

Results

Among the 2,264 patients included in the study, 28 (1.2%) presented with symptoms compatible with IBD such as diarrhea, abdominal pain, and bloody stool. The clinical

characteristics of these patients are summarized in Table 1. The mean age of the symptomatic group was 47.8 ± 11.4 years, which did not differ significantly from the overall population average. Of the 28 symptomatic patients, 18 (64.3%) were female and 10 (35.7%) were male, indicating a predominance of female patients in the symptomatic group.

The onset of symptoms occurred at a median of 5.8 months (IQR: 2.1–9.3 months) following the initiation of biological therapy.

Regarding treatment groups, 13 of the 28 patients (46.4%) with IBD-compatible symptoms were receiving ixekizumab, while 15 (53.6%) were receiving secukinumab. No statistically significant difference was found between the two drug groups in terms of IBD symptom occurrence (P=0.67).

Of the 28 symptomatic patients who were referred to gastroenterology, seven (0.3%; 7/2264) were ultimately diagnosed with definite IBD following further evaluation. Of these, four were receiving secukinumab and three were receiving ixekizumab (P=0.72).

Of the 1,120 patients treated with secukinumab, four (0.35%) were diagnosed with IBD, while among the 1,144 patients receiving ixekizumab, three (0.26%) were diagnosed.

Among those diagnosed with IBD, three had UC and four had CD. Of the UC cases, one was receiving ixekizumab and two were receiving secukinumab; among the CD cases, two were receiving ixekizumab and two were receiving secukinumab.

For patients diagnosed with IBD, the types and duration of symptoms, PsO disease duration, previous treatments, CRP and hematologic parameters, family history of PsO, and comorbidities are presented in Table 1.

The study findings indicate that in the patient population treated with IL-17 inhibitors, the rate of IBD-compatible symptoms was 1.2%, while the rate of definite IBD diagnosis was 0.3%. No significant difference in IBD development rates was found between treatment groups (secukinumab vs. ixekizumab).

Discussion

In this study, the definitive IBD rate of 0.3% detected in patients diagnosed with PsO treated with IL-17A inhibitors (secukinumab or ixekizumab) was found to be parallel to the estimated IBD prevalence rates in the general population (2%) [10,18,19]. Many meta-analyses and large-scale studies have emphasized that the risk of developing IBD under IL-17A inhibitor exposure is not significantly different from that of the general population or from that of PsO patients not using biologic agents [20-23]. A meta-analysis encompassing 16,690 patients reported 12 new IBD cases (four in patients using ixekizumab and eight in patients using secukinumab) at 60-week follow-up, reporting the general incidence rate as 2.4 and the 1-year incidence at a very low level of 0.27%, similar to non-users [21]. In another study, IBD cases were reported in 0.4% of 40,053 patients exposed to IL-17A inhibitors [23]. Eppinga et al. [18] reported the frequency of IBD in these patients as 1.6% in their retrospective study. Schreiber et al. [9], in their study covering 7,355 patients (PsO, Psoriatic Arthritis - PsA, Ankylosing Spondylitis - AS), reported the frequency of IBD events as only 0.56%. Furthermore, they concluded that these cases were generally rare among secukinumab users in the PsO subgroup. In the meta-analysis by Burisch et al. [10], the risk of developing IBD did not show a significant difference among those using IL-17A inhibitors. The doses and duration of secukinumab and ixekizumab used in these studies varied. Despite the hypothesis of a potential risk increase due to prolonged treatment duration, Yamada et al. [21] found no correlation between IL-17A inhibitor exposure duration and the development of IBD in their meta-regression analyses and reported no significant increase in rates during long-term follow-ups exceeding two years. All these findings indicate that IL-17A inhibitors do not create an adverse risk regarding gastrointestinal safety and are consistent with our retrospective data.

Nevertheless, pharmacovigilance data and case series indicate the existence of IBD cases associated with IL-17A inhibition [24]. Deng et al. [25] highlighted this potential risk by drawing attention to 268 cases linked to secukinumab and ixekizumab in data between 2015 and 2022. However, the increased IBD risk in the natural course of PsO itself should not be ignored in these cases. Meta-analysis data presented by Fu et al. [14] reported that individuals diagnosed with PsO carry a significantly high basal risk for developing both CD and UC. Therefore, the question of whether cases encountered in clinical practice are directly related to the drug effect or to this genetic and immunological predisposition in the background of PsO constitutes the main focus of discussion in the literature. The lower IBD rates observed in our study compared to those in the literature may have resulted largely from factors such as study duration, patient selection criteria, differences in geographic distribution, and follow-up intensity. Differences in retrospective data collection methods and symptom screening strategies, combined with variations in study designs and patient populations, suggest that IBD rates may vary.

Although large case series in the literature draw attention to more reports of IBD associated with secukinumab [24], no significant difference was found between the secukinumab (0.36%) and ixekizumab (0.26%) treatment groups in our study. The absolute difference between the two groups was only approximately 0.10%, and proportionally, the IBD rate in the secukinumab group was approximately 1.36 times

Table 1. Clinical characteristics of patients diagnosed with IBD.

Patient No	Age	Sex	IL-17A inhibitor used	IBD Type	Symptoms	Symptom Duration (months)	CRP (mg/L)	Leukocytes ($x10^9$/L)	Neutrophils ($x10^9$/L)	Lymphocytes ($x10^9$/L)	NLR ($x10^9$/L)	Plt ($x10^9$/L)	Previous Treatment	Family History of PsO	Comorbiditeler	PsO Duration (years)
1	29	Female	SEC	CD	Diarrhea, abdominal pain	3	12	11.8	8.2	2	4.1	400	methotrexate	yes	none	6
2	65	Male	IXS	UC	Bloody stool, fever, abdominal pain	5	14	14.5	11.2	1.5	7.5	520	methotrexate	yes	none	15
3	38	Female	IXS	CD	Diarrhea, abdominal pain	3	6	10.6	7	1.8	3.9	370	methotrexate	yes	none	9
4	61	Female	SEC	UC	abdominal pain, weight loss	6	15	13.9	10.5	1.4	7.5	480	methotrexate	yes	HT	12
5	45	Male	IXS	CD	Bloody stool, fever, abdominal pain	9	13	15.2	12.3	1.6	7.7	550	methotrexate	yes	Type 2 DM	13
6	75	Female	SEC	CD	Diarrhea, abdominal pain	3	8	11	7	1.9	4.2	390	methotrexate	yes	dementia HT	18
7	55	Female	SEC	UC	Diarrhea, abdominal pain	3	10	12.4	9	1.6	5.6	440	methotrexate	yes	HT Type 2 DM	10

Abbreviations: CPR: C-reactive protein, HT: hypertension, PLT: platelets, IXS: ixekizumab, SEC: secukinumab, UC: ulcerative Colitis , CD: Crohn's Disease .

higher than in the ixekizumab group. However, due to the very low total number of cases (N=7) and the variability of follow-up duration, the clinical significance of this difference is limited and should be interpreted with caution.

In our study, the average time to the appearance of IBD symptoms was 5.8 months. This is parallel to the literature reporting that IBD usually emerges within the first six months of treatment [26]. Reports of symptoms in earlier periods, ranging between 1.3 and four months in the literature [25, 27-29], may be due to the initial mild symptoms being ignored by patients and not reported to the physician or due to cumulative dose effects. Environmental factors such as the genetic structure of the population in our study, dietary habits, and pathogen exposures specific to the geographic region may affect the inflammatory response and tolerance mechanisms. On the other hand, IL-17A is known to have a protective role in maintaining epithelial barrier integrity in the gastrointestinal system. IL-17/IL-17RA blockade may weaken barrier function, leading to microorganism leakage and a shift in the Th1/Th2 balance [30,31]. These data suggest that rather than inducing de novo IBD, IL-17A inhibitors may trigger a "paradoxical" response that activates latent subclinical cases, as proposed by Fauny et al. [11]. Although the retrospective design of our study poses a limitation to the retrospective detection of symptom onset, long-term and careful gastrointestinal follow-up is of great importance in clinical practice.

In our study, the majority of patients exhibiting IBD symptoms were female, with a mean age of 47 years. In the literature, the sex ratios of IBD cases associated with IL-17A inhibitors are heterogeneous. While some studies present female-predominant data [22, 18, 32], other research has reported more balanced ratios or a slight male predominance [25,29]. For example, Wright et al. [22] found that 56.7% of cases were female, while Alsakarneh et al. [32] identified a female proportion of 54.9% (mean age 49.9) in a large cohort of 13,216 patients. In contrast, Deng et al. [25] reported a 1:1 ratio and a median age of 42 in pharmacovigilance data. This heterogeneity makes it difficult to establish a general rule regarding gender tendencies. The female-predominant distribution in our study may be explained by the higher tendency of female patients to seek healthcare services [33] or by the limitations of the sample size and random variations. Consequently, our data regarding the mean age appear consistent with large cohorts in the literature.

There is a lack of systematic data in the literature regarding the duration from PsO diagnosis to the initiation of IL-17A inhibitors; individual case reports generally suffice with qualitative terms such as long-term or chronic [34, 35]. Current information indicates that most IBD cases following secukinumab have had PsO for more than five years [29] and that PsO generally emerges an average of 8–12 years before IBD [14, 18]. The average PsO duration of 11.8 years detected in our study aligns perfectly with this 8–12-year window in the literature. Although this long period points to the importance of cumulative subclinical changes created by chronic inflammation in the intestinal mucosa, the lack of data still makes it difficult to define PsO disease duration as an independent risk factor for IBD development.

While the prominent symptom profile in our study was diarrhea and abdominal pain, symptoms such as bloody stools, fever, and weight loss observed in some cases are consistent with the literature. Systematic screenings and case series [23,36] define the characteristic clinical presentations of these conditions as diarrhea, abdominal pain, and bloody stools. Indeed, Deng et al. [25] reported diarrhea in 90.9%, abdominal pain in 57.6%, bloody stools in 51.5%, and fever in 36.4% of cases, reinforcing the clinical significance of the inflammatory signs observed in our series.

In our study, all 28 patients exhibiting symptoms were consulted by gastroenterology, yet only seven received a definitive IBD diagnosis. In the remaining 21 patients, clinical and laboratory findings did not indicate any pathology at an inflammatory level. In the evaluations performed by gastroenterology, noninflammatory causes such as infectious gastroenteritis, irritable bowel syndrome, or non-specific transient bowel irritation were prioritized in these cases. In these individuals, symptoms (diarrhea, abdominal pain) regressed completely within a few days, either spontaneously or with supportive treatment (oral hydration, dietary adjustment, probiotics, and antispasmodic agents). In the group that did not receive a diagnosis, IL-17A inhibitor treatment was not interrupted, and no medication change was made. Symptoms did not recur, and the long-term clinical course remained stable. This observation is consistent with the literature reporting that gastrointestinal symptoms associated with IL-17A inhibitors are generally mild and transient, resolve completely within two days to three months with supportive treatment, and mostly do not require further investigation [23, 25]. The fact that symptoms did not recur and the clinical course remained stable in the 21 undiagnosed cases suggests that the complaints may be related to non-specific, transient, or very early inflammatory phases. It should also be kept in mind that endoscopic or histological findings may not have matured yet at the onset of enteric inflammation associated with IL-17A inhibition [36]. The fact that a definitive diagnosis was made in only seven patients despite gastroenterological evaluation of all 28 patients demonstrates that diagnostic criteria were meticulously applied methodologically, thereby preventing exaggerated diagnosis rates and enhancing reliability.

In the literature, cases developing IBD after IL-17A inhibitors often have heterogeneous treatment histories, primarily involving anti-TNF, steroids, and methotrexate [18].

It has been reported that the risk of IBD in patients with prior anti-TNF experience is 8.38 times higher following the switch to IL-17A inhibitors compared to biologic-naïve patients, with symptoms emerging in as short a period as 3.67 months [9,37]. This situation reflects the cumulative effect of previous immunosuppressive treatments on the intestinal immune balance [25]. The fact that all patients diagnosed with IBD in our study had previously used methotrexate is a significant piece of clinical information. Although there is no strong evidence that methotrexate directly increases IBD risk, it is known to exert immunosuppressive effects in some cases [38,39]. This situation indicates that the cases had severe skin involvement requiring systemic treatment prior to IL-17A inhibitors. Shani et al. [40] confirmed that the presence of severe PsO requiring systemic treatment increases IBD risk by 16.03 times compared to mild cases. Therefore, the history of methotrexate in our patients should be evaluated as a clinical indicator of a high basal inflammatory load and immunological predisposition on the background of severe psoriasis rather than as a causality [25]. Prospective and multicenter studies are required to better understand this relationship.

Elevated CRP levels and leukocytosis are the most frequently observed markers in PsO patients developing IBD under IL-17A inhibitors and are crucial to follow-up. In the comprehensive analyses by Deng et al. [25], CRP elevation was detected in all cases, and a median remission duration of approximately four weeks was reported following the discontinuation of treatment. Case reports in the literature [41,34] also confirm that CRP levels rapidly return to normal upon cessation of the IL-17 inhibitor, emphasizing the diagnostic sensitivity of this biomarker. Although neutrophil-predominant leukocytosis is frequently reported as a systemic reflection of intestinal inflammation, consistent and reproducible data regarding platelet counts and the neutrophil-to-lymphocyte ratio (NLR) are not yet available [25]. The average values for leukocytes (12.77×10^9/L), neutrophils (9.44×10^9/L), NLR (5.79), and platelets (450×10^9/L) that we detected in the seven patients diagnosed with IBD in our study are in full alignment with these inflammatory trends and CRP elevations reported in the literature.

At the initial stage of our study, patients with a family history of IBD or those with a known diagnosis of irritable bowel syndrome or diverticular disease were not included in IL-17A inhibitor treatment, per the clinical protocol. However, proactive screening methods such as colonoscopic diverticulosis screening or routine fecal calprotectin measurement for asymptomatic patients, not routine practices in the literature and whose cost-effectiveness remains unproven, were not implemented in this retrospective cohort. The low IBD incidence detected in this population, which was completely asymptomatic at baseline regarding the gastrointestinal system and carried no known risk factor, reinforces the importance of careful medical history-taking and clinical inquiry.

In light of all these data, the findings we obtained demonstrate that the rates of IBD development in patients treated with IL-17A inhibitors are similar to the rates reported in the literature. The prevalence of IBD in our study population is comparable to the estimated rates in the general population [9,10]. In contrast, cases reported in the literature have shown new-onset IBD or exacerbations associated with IL-17A inhibitors; therefore, careful monitoring of gastrointestinal symptoms is especially important during the first months after starting treatment. In clinical practice, when planning IL-17A inhibitor therapy for PsO, the patient's history regarding IBD should be meticulously evaluated. Current evidence suggests strategies such as proactive screening or calprotectin measurement. Future studies require detailed monitoring of IBD-related indications and side effects to clarify potential differences that may arise with long-term use and new IL-17A-targeted drugs.

Limitations

Due to the retrospective, single-center design, the generalizability of our results is limited and insufficient to definitively establish the effect of IL-17A inhibitors on IBD risk. The lack of clarity regarding individual patient follow-up duration restricted the calculation of incidence and relative risk. Additionally, the relatively small number of patients reduces the statistical power. Due to the retrospective design, advanced tests such as calprotectin and histological examination were not routinely performed in all patients and were applied only to cases with suspected IBD. Therefore, homogeneous data covering the entire cohort could not be included in the analysis.

Conclusion

In this study, of 2,264 PsO patients treated with IL-17A inhibitors (secukinumab and ixekizumab), the rate of symptoms compatible with IBD was found to be 1.2%, while the rate of definitive IBD diagnosis was 0.3%. No significant difference was found between the secukinumab (0.35%) and ixekizumab (0.26%) groups in terms of IBD development. In most cases diagnosed with IBD, symptoms emerged within the first months of treatment, and the clinical findings are consistent with the literature. Our findings suggest that IL-17A inhibitors do not significantly increase the risk of IBD in psoriasis patients and that the rates remain at levels similar to those reported in the general population. Nevertheless, careful monitoring of gastrointestinal symptoms, which may arise particularly during the initial months of treatment, is of clinical importance.

References

1. Bianchi E, Rogge L. The IL-23/IL-17 pathway in human chronic inflammatory diseases: new insight from genetics and targeted therapies. *Genes Immun*. 2019;20(5):415-425. DOİ: 10.1038/s41435-019-0067-y
2. Onishi RM, Gaffen SL. Interleukin-17 and its target genes: mechanisms of interleukin-17 function in disease. *Immunology*. 2010;129(3):311-321. DOI: 10.1111/j.1365-2567.2009.03240.x
3. Gálvez J. Role of Th17 cells in the pathogenesis of human IBD. *ISRN Inflamm*. 2014;2014:928461. DOI: 10.1155/2014/928461
4. Ali AK, Torosian A, Porter C, Bloomfeld RS, Feldman SR. New onset inflammatory bowel disease in patient treated with secukinumab: case report and review of literature. *Dermatol Ther*. 2021;34(6):e15151. Doi:10.1111/dth.15151
5. Fieldhouse KA, Ukaibe S, Crowley EL, Khanna R, O'Toole A, Gooderham M. JInflammatory bowel disease in patients with psoriasis treated with interleukin-17 inhibitors. *Drugs Context*. 2020;9:2020-7-2. DOI: 10.7573/dic.2020-2-1
6. Achufusi TG, Harnee PS, Rawlins S. A rare case of new-onset ulcerative colitis following initiation of secukinumab. *Case Rep Med*. 2019;2019:2975631. DOI: 10.1155/2019/2975631
7. Ehrlich D, Jamaluddin N, Pisegna J, Padua D. A challenging case of severe ulcerative colitis following the initiation of secukinumab for ankylosing spondylitis. *Case Rep Gastrointest Med*. 2018;2018:9679287. DOI: 10.1155/2018/9679287
8. Wang J, Bhatia A, Cleveland NK, et al. Rapid onset of inflammatory bowel disease after receiving secukinumab infusion. *ACG Case Rep J*. 2018;5:e56. DOI: 10.14309/crj.2018.56
9. Schreiber S, Colombel JF, Feagan BG, et al. Incidence rates of inflammatory bowel disease in patients with psoriasis, psoriatic arthritis and ankylosing spondylitis treated with secukinumab: a retrospective analysis of pooled data from 21 clinical trials. *Ann Rheum Dis*. 2019;78(4):473-479. DOI: 10.1136/annrheumdis-2018-214273
10. Burisch J, Eigner W, Schreiber S, et al. Risk for development of inflammatory bowel disease under inhibition of interleukin 17: a systematic review and meta-analysis. *PLoS One*. 2020;15(5):e0233781. DOI: 10.1371/journal.pone.0233781
11. Fauny M, Moulin D, D'Amico F, et al. Paradoxical gastrointestinal effects of interleukin-17 blockers. *Ann Rheum Dis*. 2020;79(9):1132-1138.DOI:10.1136/annrheumdis-2020-217927
12. Petitpain N, D'amico F, Yelehe-Okouma M, et al. IL-17 inhibitors and inflammatory bowel diseases: a postmarketing study in VigiBase. *Clin Pharmacol Ther*. 2021;110(1):159-168. DOI: 10.1002/cpt.2155
13. Goepfert A, Lehmann S, Wirth E, Rondeau JM. The human IL-17A/F heterodimer: a two-faced cytokine with unique receptor recognition properties. *Sci Rep*. 2017;7(1):8906. DOI: 10.1038/s41598-017-08360-9
14. Fu Y, Lee CH, Chi CC. Association of psoriasis with inflammatory bowel disease: a systematic review and meta-analysis. *JAMA Dermatol*. 2018;154(12):1417-1423. DOI: 10.1001/jamadermatol.2018.3631
15. Targan SR, Feagan B, Vermeire S, et al. A randomized, double-blind, placebo-controlled phase 2 study of brodalumab in patients with moderate-to-severe Crohn's disease. *Am J Gastroenterol*. 2016;111(11):1599-1607. DOI: 10.1038/ajg.2016.298
16. Hueber W, Sands BE, Lewitzky S, et al. Secukinumab, a human anti-IL-17A monoclonal antibody, for moderate to severe Crohn's disease: unexpected results of a randomized, double-blind placebo-controlled trial. *Gut*. 2012;61(12):1693-1700. DOI: 10.1136/gutjnl-2011-301668
17. Kobayashi T, Okamoto S, Hisamatsu T, et al. IL-23 differentially regulates the Th1/Th17 balance in ulcerative colitis and Crohn's disease. *Gut*. 2008;57(12):1682-1689. DOI: 10.1136/gut.2007.135053
18. Eppinga H, Poortinga S, Thio HB, et al. Prevalence and phenotype of concurrent psoriasis and inflammatory bowel disease. *Inflamm Bowel Dis*. 2017;23(10):1783-1789. DOI: 10.1097/MIB.0000000000001169
19. GBD 2017 Inflammatory Bowel Disease Collaborators. The global, regional, and national burden of inflammatory bowel disease in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet Gastroenterol Hepatol*. 2020;5(1):17-30. DOI: 10.1016/S2468-1253(19)30333-4
20. Alinaghi F, Tekin HG, Burisch J, Wu JJ, Thyssen JP, Egeberg A. Global prevalence and bidirectional association between psoriasis and inflammatory bowel disease a systematic review and meta-analysis. *J Crohns Colitis*. 2020;14(3):351-360.DOI:10.1093/ecco-jcc/jjz152
21. Yamada A, Wang J, Komaki Y, Komaki F, Micic D, Sakuraba A. Risk of new onset IBD with the use of anti-interleukin-17 agents: a systematic review and meta-analysis. *Aliment Pharmacol Ther*. 2019;50(4):373-385. DOI: 10.1111/apt.15397
22. Wright S, Alloo A, Strunk A, Garg A. Real-world risk of new-onset inflammatory bowel disease among patients with psoriasis exposed to interleukin 17 inhibitors. *J Am Acad Dermatol*. 2020;83(2):382-387. DOI: 10.1016/j.jaad.2020.04.010
23. Caron B, Jouzeau JY, Miossec P, et al. Gastroenterological safety of IL-17 inhibitors: a systematic literature review. *Expert Opin Drug Saf*. 2022;21(2):223-239. DOI: 10.1080/14740338.2021.1960981
24. Onac IA, Clarke BD, Tacu C. Secukinumab as a potential trigger of inflammatory bowel disease in ankylosing spondylitis or psoriatic arthritis patients. *Rheumatology (Oxford)*. 2021;60(11):5233-5238. DOI: 10.1093/rheumatology/keab193
25. Deng Z, Wang S, Wu C, Wang C. IL-17 inhibitor-associated inflammatory bowel disease: a study based on literature and database analysis. *Front Pharmacol*. 2023;14:1124628. doi:10.3389/fphar.2023.1124628
26. Cepero-Jimenez CM, Santiago-Rivera L. New-onset ulcerative colitis associated to ixekizumab. *Am J Gastroenterol*. 2024;119(12 Suppl):S28. DOI: 10.14309/01.ajg.0001082952.32829.7b
27. Masic N, Anjie SI, Sungur, R. New-onset inflammatory bowel disease is uncommon in patients with psoriasis, psoriatic arthritis and spondylarthritis treated with secukinumab: a retrospective cohort study in a tertiary centre. *J Crohns Colitis*. 2024;18(Suppl 1):i2230. Doi:10.1093/ecco-jcc/jjad212.1250
28. Letarouilly JG, Pham T, Pierache A. New-onset inflammatory bowel diseases among IL-17 inhibitor-treated patients: results from the case–control MISSIL study. *Rheumatology (Oxford)*. 2022;61(7):2848-2855. DOI: 10.1093/rheumatology/keab819
29. Li R, Lei H, Wang C, Liu X. Clinical features, treatment, and prognosis of secukinumab-induced inflammatory bowel disease. *Eur J Med Res*. 2025;30(1):37. DOI: 10.1186/s40001-025-02295-y

30. Maxwell JR, Yadav R, Rossi RJ. IL-18 bridges innate and adaptive immunity through IFN-γ and the CD134 pathway. *J Immunol*. 2006;177(1):234-245. DOI: 10.4049/jimmunol.177.1.234
31. Ogawa A, Andoh A, Araki Y, Bamba T, Fujiyama Y. Neutralization of interleukin-17 aggravates dextran sulfate sodium-induced colitis in mice. *Clin Immunol*. 2004;110(1):55-62. DOI: 10.1016/j.clim.2003.09.013
32. Alsakarneh S, Al Ta'ani O, Aburumman R, Mikhail I, Hashash JG, Farraye F. Risk of de novo inflammatory bowel disease in patients with psoriasis and psoriatic arthritis treated with IL-17A inhibitors: a population-based study. *Aliment Pharmacol Ther*. 2025;62(3):321-332. DOI: 10.1111/apt.70139
33. Colombo D, Cassano N, Bellia G, Vena GA. World Journal of. *World J*. 2014;3(3):36-44. doi:10.5314/wjd.v3.i3.36
34. Dai H, Xu C, Wang Y. Secukinumab-induced Crohn's disease in a psoriasis patient: a case report highlighting paradoxical reactions to IL-17 inhibition. *Front Immunol*. 2025;16:1628461. DOI: 10.3389/fimmu.2025.1628461
35. Bratborska A, Jałowska M, Bowszyc-Dmochowska M, Kowalczyk MJ, Adamska K. Misfortunes never come alone: melanoma and ulcerative colitis after biologic therapy in a psoriatic patient - a case report and literature review. *Front Immunol*. 2025; 16:1651517. DOI: 10.3389/fimmu.2025.1651517
36. Grümme L, Dombret S, Knösel T, Skapenko A, Schulze-Koops H. Colitis induced by IL-17A inhibitors. *Clin J Gastroenterol*. 2024;17(2):263-270. DOI: 10.1007/s12328-023-01893-9
37. Albayrak F, Gür M, Karataş A, Koca SS, Kısacık B. Is the use of secukinumab after anti-TNF therapy greater than expected for the risk of developing inflammatory bowel disease? *Reumatol Clin*. 2024;20(3):123-127. DOI: 10.1016/j.reumae.2023 .11.002
38. Orzan OA, Ţieranu CG, Olteanu AO, et al. An insight on the possible association between inflammatory bowel disease and biologic therapy with IL-17 inhibitors in psoriasis patients. *Pharmaceutics*. 2023;15(8):2171. DOI: 10.3390/pharmaceutics15082171
39. Nehring P, Przybyłkowski A. Is psoriasis treatment a risk factor for inflammatory bowel disease? *Pharm Med*. 2020;34(4):257-262. DOI: 10.1007/s40290-020-00340-1
40. Shani U, Ben-Shabat N, Qassem R. The association between psoriasis, psoriasis severity, and inflammatory bowel disease: a population-based analysis. *Ther Adv Gastroenterol*. 2024;17: 17562848241227037. DOI: 10.1177/17562848241227037
41. Smith MK, Pai RK, Russo PA, et al. Crohn's-like disease in a patient exposed to anti-interleukin-17 blockade (ixekizumab) for the treatment of chronic plaque psoriasis: a case report. *BMC Gastroenterol*. 2019;19(1):1-6. DOI: 10.1186/s12876-019-1067-0