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The onset of ulcerative colitis during treatment with secukinumab: can anti-IL-17A be a trigger for inflammatory bowel disease?

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Dear Editor,

Secukinumab is a monoclonal antibody that acts specifically on interleukin 17A (IL-17A) and is used for the treatment of plaque psoriasis, psoriatic arthritis and ankylosing spondylitis (1). IL-17A is a pro-inflammatory cytokine raised in the intestinal mucosa of patients with inflammatory bowel disease (IBD). However, this is paradoxical, as blocking the IL-17 pathway using secukinumab is not associated with a reduction in bowel inflammation. In fact, it seems to make it worse (2,3). Furthermore, IL-17 seems to act as a protector against inflammation, contributing to the inhibition of the Th1 response and maintaining the integrity of the epithelial barrier of the enterocyte and intestinal homeostasis (2). Therefore, caution is needed when administering secukinumab to IBD patients, as it could result in outbreaks of activity (4). However, it has not been identified as a trigger of IBD that was not already present. Here we describe a case observed in our unit.

Case report

The case was a 42-year-old male with psoriatic arthritis who was under treatment with secukinumab as he had not responded to methotrexate. There was nothing of note in his medical history, except that his mother had been diagnosed with ulcerative colitis. Around three weeks after the treatment began, the patient started to produce

diarrheal stools and rectal bleeding. A colonoscopy was performed and inflammatory changes were observed in the mucosa, from the anal margin to the splenic angle, with a histology compatible with ulcerative colitis. As a result, treatment with secukinumab was suspended and treatment began with corticosteroids and golimumab.

Conclusion

Recently, several cases have been reported that resemble ours, i.e., the onset of IBD in patients with rheumatic disorders treated with secukinumab during the first weeks after treatment initiation. This is summarized in table 1. Thus, the drug could be triggering an outbreak of activity and also activates cases of latent illness in genetically predisposed patients (our case had a family history). Therefore, we should consider using other safer therapeutic options for the treatment of these entities (1,5).

References

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Table 1. Secukinumab and the onset of IBD. A review of the available literature

Publication	*Gender*	*Age*	*Underlying illness*	*IBD*	*Secukinumab dosage**	*Time after first dose when illness appeared*
Fobelo Lozano MJ et al. 2018 (1)	Female	19	Plaque psoriasis	Crohn's disease	300 mg/week (induction) 300 mg/month (maintenance)	7-8 weeks
Fobelo Lozano MJ et al. 2018 (1)	Male	60	Ankylosing spondylitis	Ulcerative colitis	150 mg/week (induction) 150 mg/month (maintenance)	2-3 weeks
Ehrlich D et al. 2018 (5)	Male	42	Ankylosing spondylitis	Ulcerative colitis	Not specified	6-7 weeks
Wang J et al. 2018 (4)	Female	41	Plaque psoriasis	Indeterminate colitis	Not specified	1-2 weeks
†	Male	42	Psoriatic arthritis	Ulcerative colitis	150 mg/week (induction) 150 mg/month (maintenance)	3-4 weeks

Cases to date that describe the onset of IBD in patients with rheumatic disorders treated with secukinumab following its commercialization. *Induction was performed by the administration of the dosage according to pathology in weeks 0, 1, 2, 3 and 4, and then monthly during the maintenance phase. †It describes the case presented in this article.