data.[2,3,5] Both conditions are relatively uncommon with similar prevalence. They are more common in White and Asian female patients aged between 40 and 70 years.

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Brodalumab success in patients with moderate-to-severe psoriasis who failed previous interleukin-17A inhibitors

To the Editor: The interleukin-17 (IL17) signaling pathway plays a pivotal role in the pathogenesis of psoriasis. While several studies investigated the efficacy and safety of switching between IL17A inhibitors, data on switching from IL17A inhibitors to the IL17 receptor A (IL17RA) antagonist brodalumab remain limited.

Clinical outcomes in patients switched from secukinumab or ixekizumab to brodalumab have been reported in 3 studies.[1-3] Gasslitter et al[1] first reported that 50% (3/7) and 67% (2/3) of patients who failed secukinumab and ixekizumab, respectively, achieved 75% improvement in Psoriasis Area and Severity Index (PASI75) after 12 weeks of treatment with brodalumab. In an open-label study that included 39 patients with moderate to severe psoriasis who had failed treatment with an IL17A inhibitor, 69% of patients achieved PASI75 after 16 weeks of brodalumab treatment.[2] Consistent with these data, Kromer et al[3] found that approximately 48% of psoriasis patients (11/23) who previously failed treatment with an IL17A inhibitor achieved PASI75 after 12 weeks of brodalumab treatment.

We conducted a Canadian multicenter retrospective study of 47 patients with chronic plaque psoriasis who were treated with brodalumab after discontinuation of secukinumab or ixekizumab because of nonresponse (44/47), response optimization (2/47), or an adverse event (1/47). Primary nonresponders were subjects who did not achieve PASI75 at weeks 12 to 16 on IL17A inhibitors. Secondary nonresponders were patients who achieved PASI75 at weeks 12 to 16 but lost PASI75 response thereafter. The primary endpoint for this study was PASI100 after 16 weeks of brodalumab treatment, whereas PASI75 and PASI90 at week 16 were secondary endpoints.

Patient demographics and clinical characteristics of study participants are summarized in Table I. Most patients were male (66%) with a mean age of 52 years. In addition, 42.5% of patients had psoriatic arthritis at baseline. Primary or secondary nonresponse were the main reasons for discontinuation

Table I. Patient demographics and clinical characteristics at baseline and after 16 weeks of treatment with brodalumab

Demographic and clinical characteristic	Value
Mean age, y ± SD	51.8 ± 14.7
Sex, n/N (%)	
Male	31/47 (66)
Female	16/47 (34)
Mean body mass index, kg/m^2 ± SD*	31.3 ± 7.4
Psoriatic arthritis at baseline, n/N (%)	20/47 (42.5)
Failed biologics before brodalumab, n, mean ± SD	2.9 ± 1.4
Previously discontinued treatment(s), n/N (%)	
Secukinumab†	14/47 (29.8)
Ixekizumab‡	13/47 (27.7)
Secukinumab and ixekizumab§	20/47 (42.5)
Months treated with secukinumab, mean ± SD	15.1 ± 10.5
Months treated with ixekizumab, mean ± SD	17 ± 17
PASI score before initiation of brodalumab, mean ± SD	10.1 ± 7.6
Efficacy	
Brodalumab responders at week 16, n/N (%)	
PASI75	29/47 (61.7)
PASI90	22/47 (46.8)
PASI100	20/47 (42.5)
Brodalumab PASI90 responders stratified by number of previous biologics, n/N (%)	
1 previous biologic	3/7 (43)
2 previous biologics	5/13 (38.5)
3 previous biologics	7/12 (58.3)
4 previous biologics	4/9 (44.4)
5 previous biologics	1/3 (33.3)
6 previous biologics	2/3 (66.6)

PASI, Psoriasis Area and Severity Index.
*Body mass index–specific values were not reported for 7 patients, 5 of which had normal body mass indices.
†Of the 14 patients, 2 were primary nonresponders, 11 were secondary nonresponders, and 1 discontinued because of adverse events.
‡Of the 13 patients, 2 were primary nonresponders, 9 were secondary nonresponders, and 2 were switched to brodalumab to optimize response (physician's global assessment score of 3).
§For secukinumab, 10 were primary nonresponders, and 10 were secondary nonresponders. For ixekizumab, 6 were primary nonresponders, and 14 were secondary nonresponders.

of IL17A inhibitors (Table I). Brodalumab was discontinued before week 16 in 3 patients (6.4%) because of primary nonresponse (1/3), cystitis (1/3), or severe psoriasis flare (1/3).

Of the 47 patients who stopped previous IL17A inhibitors, 20 (42.5%) achieved PASI100 with brodalumab at week 16 (Fig 1). PASI90 and PASI75 at week

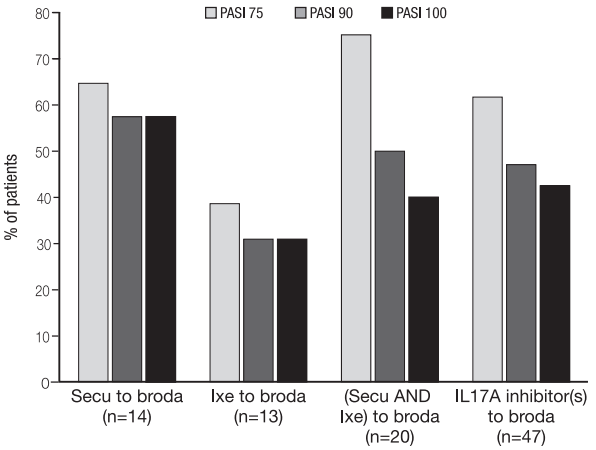

Fig 1. Proportion of patients who discontinued an interleukin-17A (IL17A) inhibitor and achieved Psoriasis Area and Severity Index (PASI) improvements of 75%, 90%, and 100% with brodalumab at week 16. Fourteen patients discontinued secukinumab, 13 patients discontinued ixekizumab, and 20 patients discontinued both agents before receiving brodalumab.

16 was achieved by 22 (46.8%) and 29 (61.7%) patients, respectively. A significant proportion of PASI90 responders were observed in patients who had received 3 previous biologics (Table I). Notably, a higher proportion of patients switched from secukinumab achieved PASI100 on brodalumab (8/14; 57%) compared with those switched from ixekizumab (4/13; 31%) (Fig 1), which is in line with previously published data.[3] In addition, subgroup analysis by type of nonresponse showed that a higher proportion of secondary nonresponders to IL17A inhibitors achieved PASI100 on brodalumab (secukinumab 13/21 [62%]; ixekizumab 10/23 [43.4%]) compared with those who were primary nonresponders (secukinumab 3/12 [25%]; ixekizumab 1/8 [12.5%]).

This is the largest study conducted to date on the efficacy of brodalumab in IL17A inhibitor nonresponders. We postulate that differential efficacy with brodalumab could be attributed to IL17RA antagonism, resulting in a more complete suppression of pathogenic signaling. Collectively, these data suggest that brodalumab can be successfully used in IL17A inhibitor nonresponders, with potentially higher efficacy in secukinumab versus ixekizumab nonresponders.

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Conflicts of interest

Dr Yeung has been a speaker, consultant, and investigator for AbbVie, Allergan, Amgen, Astellas, Bausch Health, Boehringer Ingelheim, Celgene, Centocor, Coherus, Dermira, Eli Lilly, Forward, Galderma, GSK, Janssen, Leo, Medimmune, Merck, Novartis, Pfizer, Regeneron, Roche, Sanofi Genzyme, Takeda, UCB, Valeant, and Xenon. Dr Vender has received grants/research support from Abbvie, Amgen, Centocor, Dermira, Dermavant, Galderma, GSK, Leo, Lilly, Takeda, Novartis, Merck, Pfizer, Regeneron, and UCB. Dr Vender has also received honoraria and consulting fees from Abbvie, Amgen, Janssen, Galderma, GSK, Leo, Lilly, Novartis, Pfizer, Bausch-Health, Actelion, Celgene, Cipher, and UCB. Dr Turchin has received honoraria and consulting fees from Abbvie, Bausch Health, Celgene, Amgen, Eli Lilly, Janssen, LeoPharma, Novartis, and UCB. Dr Turchin has also been involved in clinical trials sponsored by Abbvie, Amgen, Arcutis, Boehringer Ingelheim, Celgene, Eli Lilly, Janssen, LeoPharma, and Novartis. Dr Shukla has received honoraria and consulting fees from Abbvie, Amgen, Bausch, Galderma, Janssen, Leo, Lily, Novartis, Pfizer, Sanofi, and UCB. Dr Hong is a researcher/consultant/advisor for AbbVie, Amgen, Arcutis, Bausch Health, Boehringer Ingleheim, Bristol-Meyer Squibb, Celgene, Dermira, Dermavant, DS Biopharma, Galderma, GlaxoSmithKline, Janssen, LEO Pharma, Lilly, MedImmune, Novartis, Pfizer, Regeneron, Roche, Sanofi Genzyme, and UCB. Dr Maari is an investigator, Advisory Board Member, Speaker, Consultant for, and received honoraria or grants from Abbvie, UCB, Boehringer Ingelheim, Celgene, Eli Lilly, Galderma, Leo Pharma, GSK-Stiefel, Janssen, Novartis, Bausch, and Pfizer. Dr Lansang has received honoraria and consulting fees from AbbVie, Amgen, Bausch, Celgene, Galderma, Janssen, Lilly, Novartis, Pfizer, Sanofi, UCB, and Valeant.

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Long-term efficacy and safety outcomes of lenalidomide for cutaneous lupus erythematosus: A multicenter retrospective observational study of 40 patients

To the Editor: Small case series have suggested that lenalidomide may be a promising therapeutic option for severe cutaneous lupus erythematosus (CLE).[1-3] The aims of this study were to report the long-term efficacy and safety profile of lenalidomide in patients with CLE with a focus on patients with associated systemic lupus erythematosus (SLE) and potential factors associated with complete response (CR).

This multicenter retrospective observational case series enrolled patients with CLE who received lenalidomide after failure of hydroxychloroquine and ≥1 second-line systemic treatment.

Clinical efficacy was assessed with the CLE Disease and Severity Index Activity[4] (CLASI-A) score at baseline, at first evaluation scheduled after 1 or 3 months, and every 6 months thereafter. Cutaneous response was defined as follows: minimal response was defined by a 4-point or 20% decrease in CLASI-A score, partial response (PR) by an improvement of ≥50%, and CR by a CLASI-A score of 0. Occurrence of relapse, doses at which they occurred, and SLE flares were recorded.