

Secukinumab drug survival in patients with psoriasis: A multicenter, real-world, retrospective study



To the Editor: Secukinumab, an interleukin 17A inhibitor approved for the treatment of moderate-to-severe plaque psoriasis, has shown high levels of clinical efficacy and a favorable safety profile in both short-term and long-term clinical trials. However, in some studies in the real-life setting, secukinumab showed a 73%-78% drug survival at 12 months, shorter than that of other biologic agents, even among biologic-naive patients, and loss of effectiveness was the most common reason for discontinuation.¹⁻⁴

To evaluate drug survival of secukinumab in a daily practice setting, we retrospectively evaluated all psoriatic patients starting secukinumab during January 2016-February 2017 at 11 centers in 3 European countries and followed them until February 2018 (potentially with at least 1 year of observation period).

Failure was defined as stopping secukinumab definitively or switching to a different therapy. Drug survival was analyzed using Kaplan–Meier methodology.

A total of 330 patients were included. Their median \pm standard deviation age was 51.9 ± 14.6 years, 68% were male, and 19.6% were obese (body mass index [BMI] ≥ 30 kg/m²); 21.5% of patients had a diagnosis of psoriatic arthritis, and 52.4% were biologic-experienced (24.5% with ≥ 2 prior biologics). All patients were treated with the 300-mg label dose, and there was no dose adjustment throughout the treatment. Concomitant systemic therapy was used in 12% of patients (mostly methotrexate).

Overall drug survival of secukinumab was 83% after 12 months and 78.8% after 18 months (Fig 1, A). The dropout rate at 18 months was 20%: 12.4% of patients were lost to follow-up, 6.7% of patients stopped because of drug ineffectiveness, 0.3% of patients discontinued because of adverse events, and 0.6% of patients decided to quit the drug.

Drug survival rates at 12 and 18 months were lower for biologic-experienced than biologic-naive patients (Fig 1, B) and lower in obese than nonobese patients. Patients treated with concomitant systemic therapies also showed a lower drug survival rate than those treated with secukinumab monotherapy at 12 months (79.5% vs 94.2%, respectively) and 18 months (53.7% vs 81.8%, respectively).

Univariate Cox regression analysis of drug survival determinants showed that prior use of >1

biologic and obesity at baseline were associated with decreased secukinumab drug survival ($P < .05$ or 95% confidence interval >1). The other variables evaluated (sex, age, presence of psoriatic arthritis, or baseline Psoriasis Area and Severity Index) were not found to be significantly associated with drug survival.

In this study, overall drug survival of secukinumab was higher than previously described.^{3,4} Although the reasons for the previously reported low secukinumab drug survival remains unclear, small patient cohorts with a high portion of biologic-experienced patients,^{1,3} who usually represent a difficult-to-treat population, could in part explain these results. Consistent with other studies, being biologic-experienced was associated with shorter drug survival.¹ Lower secukinumab drug survival was seen in obese patients (BMI ≥ 30 kg/m²), whereas another study identified body weight, not BMI, as a factor negatively affecting secukinumab drug survival. Nevertheless, BMI was also associated with poorer treatment response in both real-life and trial settings.^{2,5}

In our experience, drug survival of secukinumab is not as low as initially reported. These findings are likely affected by the high rate of patients naive to biologic therapy (47.6%) and by the dropout rate (17% at 12 months and 20% at 18 months) in line or slightly lower than some recent real-life studies.⁵ Our study indicates the durability of secukinumab therapy in treating plaque psoriasis patients in the real-world setting, with relatively lower drug survival in biologic-experienced and obese patients.

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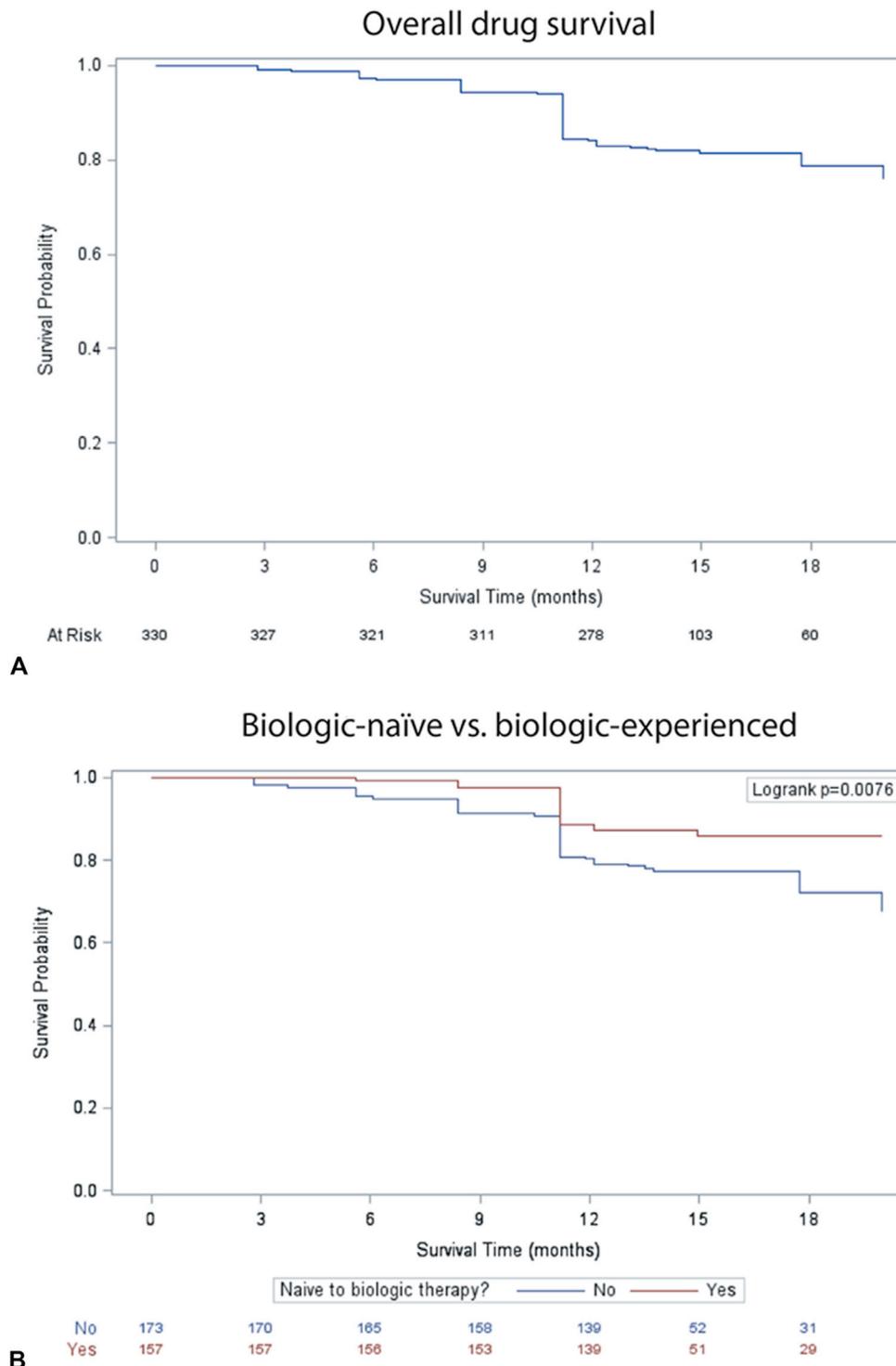


Fig 1. Kaplan–Meier plots of secukinumab drug survival in psoriasis patient population. Overall survival rate (**A**) and survival rate in bio-naïve versus biologic-experienced patients (**B**). Graphs show discontinuation due to any cause.

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Conflict of interest: Dr Torres has served as a scientific consultant, speaker, and clinical study investigator for AbbVie, Amgen, Boehringer Ingelheim, Biogen, Celgene, Janssen, LEO-Pharma, Eli-Lilly, MSD, Novartis, Pfizer, Samsung Bioepis, and Sanofi. Dr Conrad has served as a scientific consultant, speaker, and clinical study investigator for AbbVie, Actelion, Amgen, Celgene, Janssen, LEO Pharma, Lilly, MSD, Novartis, and Pfizer. Dr Ferreira served as scientific consultant, speaker, and clinical study investigator for AbbVie, Janssen, LEO-Pharma, Eli-Lilly, Novartis, and Pfizer. Dr Leite served as scientific consultant, speaker, and clinical study investigator for AbbVie, Janssen, Eli-Lilly, and Novartis. Dr Mendes-Bastos has served as a scientific consultant, speaker, and clinical study investigator for AbbVie, Pfizer, Janssen, LEO Pharma, Novartis, Sanofi, Teva, Bayer, and L'Oreal. Dr Piaserico has served as scientific consultant, speaker, and clinical study investigator for AbbVie, Celgene, Galderm, Janssen, LEO Pharma, Lilly, MSD, Novartis, and Pfizer. Dr Chiricozzi has served as scientific consultant, speaker, and clinical study investigator for AbbVie, Biogen, Eli Lilly and Company, Janssen, Leo Pharma, Novartis, UCB Pharma, and Sanofi Genzyme. Dr Balato, Dr Conti, Dr Dapavo, Dr Gaiani, Dr Malagoli, Dr Megna, Ms Valério, Dr Messina, Dr Nidegger, Dr Odorici, Dr Prignano, Dr Ribero, Dr Ricceri, and Dr Tonini have no conflicts of interest to disclose.

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Tracking changes in nailfold capillaries during dermatomyositis treatment



To the Editor: The clinical features of dermatomyositis are believed to be due in part to small vessel vasculopathy and perivascular inflammation, which leads to a reduction in capillary density with compensatory dilatation of the remaining capillaries.¹ In the skin, a manifestation of this is easily measurable is changes in capillary nailfolds. Indeed, a retrospective study of 92 juvenile dermatomyositis patients showed that nailfold capillary density is a marker of not just skin damage but also muscle damage and, therefore, appears to reflect the degree of systemic blood vessel alterations.² Another study of 60 patients with juvenile dermatomyositis showed that low nailfold capillary density is associated with impaired pulmonary function tests.³ The question remains if these changes in adult dermatomyositis are permanent and represent a persistent measure of overall disease-induced damage, or if on therapy, they are reversible, suggesting they are more a measure of disease activity. Our objective was to assess if these capillary changes reverse with the use of immunosuppressant therapy and if the rate or extent of reversal varies between therapeutic options.

We performed a retrospective chart review of all patients seen in Massachusetts General Hospital's combined Rheumatology-Dermatology clinic during November 2012-June 2018. Nailfold capillaroscopy exam of all fingers was performed by the same dermatologist with the same optical dermatoscope at each visit. The following parameters of periungual capillaries were assessed: dilatation, dropout, and reangiogenesis. The presence of the aforementioned parameters in ≥ 2 fingers was considered remarkable.