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Biologic Treatment of 4 HIV-Positive Patients: A Case Series and Literature Review

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Background:

The management of psoriatic disease in human immunodeficiency virus (HIV)-positive patients is challenging. Psoriasis in HIV-positive patients is often severe, progressive, and resistant to first- and second-line therapies, including topical treatments, phototherapy, highly active antiretroviral therapy (HAART), and oral retinoids. Other systemic agents used to treat psoriasis, such as methotrexate and cyclosporine, are immunosuppressants and thus many dermatologists may not feel comfortable prescribing them to HIV-positive patients who are already immunocompromised. Biologic agents, which target specific aspects of overactive immune pathways in psoriasis, have revolutionized the management of moderate-to-severe psoriasis. However, data is limited regarding their safety and efficacy in HIV-positive patients. Objective: Report four cases of HIV-positive patients managed on biologic therapy and summarize the cases of psoriasis in HIV-positive patients managed on biologic therapy that have been published in dermatologic literature to date. Methods: We searched PubMed and Embase databases using the terms HIV and psoriasis or HIV and psoriatic arthritis combined with one of the eleven biologics currently approved for treating psoriasis. Results: We identified 48 cases of anti-psoriasis biologic therapy (including adalimumab, infliximab, etanercept, ustekinumab, and guselkumab) in HIV-positive patients and added four. While data is limited, the evidence available suggests biologic agents are safe and efficacious in moderate-to-severe psoriasis and may even have a favorable effect on CD4 and HIV viral counts when used with concomitant HAART. Conclusion: Further research would be helpful to establish practical guidelines for the use of anti-psoriasis biologic therapy in the HIV population, including that of newer agents.

Keywords

psoriasis; psoriatic arthritis; biologic treatment; human immunodeficiency virus (HIV); acute immunodeficiency virus (AIDs)

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Introduction

In the U.S. the prevalence of psoriasis in the HIV population is estimated to be between 1 and 3%, which is comparable to that reported in the general population.[1] While HIV may not be an independent trigger for the development of psoriasis, it can be an aggravating factor in afflicted patients.[2] For example, in patients with pre-existing psoriasis, their skin disease often flares following initial infection with HIV and may worsen as their HIV disease burden progresses.[3] Psoriasis in HIV-positive patients is especially difficult to manage, as their disease is often severe, progressive, and resistant to recommended first- and second-line therapies, such as topical treatments, phototherapy, highly active antiretroviral therapy (HAART), and oral retinoids.[2,4] Other systemic agents used in psoriasis management such as methotrexate and cyclosporine, are immunosuppressants, which many providers are reluctant to use in HIV-positive patients who are already immunocompromised. Biologic agents, which target the overactive immune pathways in psoriasis, are widely used to treat moderate-to-severe disease. However data are limited regarding the efficacy and safety of biologic agents in treating HIV-positive psoriasis patients as these individuals are often excluded from clinical trials.[5]

Here, we provide a case series of four HIV-positive psoriasis patients successfully managed with biologic therapy using adalimumab, ustekinumab, or etanercept (Table 1). We also offer an up-to-date summary of all reported cases of HIV-positive psoriasis patients on biologic therapy and review the efficacy and safety data from these studies.

Methods

A thorough search of PubMed and Embase databases was completed using the search terms ‘HIV’ and ‘psoriasis’ or ‘psoriatic arthritis’ and ‘biologic’ or ‘infliximab’ or ‘adalimumab’ or ‘etanercept’ or ‘ustekinumab’ or ‘ixekizumab’ or ‘secukinumab’ or ‘brodalumab’ or ‘guselkumab’ or ‘certolizumab pegol’ or ‘tildrakizumab’ or ‘risankizumab.’ Our search was limited to English-language articles and those published prior to March 19, 2020. We manually identified relevant articles, excluding duplicates. In total, we identified 19 case reports or case series and one retrospective review regarding the use of biologic therapy in treating psoriasis in HIV-positive patients (Table 2). An additional eight reviews on psoriasis or biologic therapy in the HIV population were also included.

Case 1

Patient 1 is a 42-year-old male with a long-standing history of severe generalized plaque psoriasis. He acquired HIV prior to developing psoriasis and was stable on HAART on presentation. When his psoriasis was initially diagnosed, he was started on etanercept with good results. He began to experience a loss of efficacy after three years of treatment, so he switched to adalimumab with much improvement. He presented to our clinic while on adalimumab. His HIV viral load at that time was undetectable and his CD4 count was 1,300 cells/μL. His adalimumab was continued, but after more than two years of treatment his psoriasis began to flare, resulting in thick bilateral lower extremity plaques. Following several weeks of poorly controlled disease despite adalimumab and maximal topical therapy, the patient transitioned to ustekinumab therapy 90 mg every 12 weeks.

Significant improvement was noted at week eight, and his bilateral lower extremity plaques cleared completely. He has now been treated with ustekinumab for over 18 months and has maintained disease remission with no adverse events. His most recent CD4 count was 1,652 cells/μL and his HIV viral load remains undetectable.

Case 2

Patient 2 is a 58-year-old male who presented to our clinic with generalized, erythematous plaques covering over 75% of his body surface area. His medical history included alcohol abuse disorder, chronic Hepatitis B, and HIV infection. He is an HIV elite controller,[6] with his HIV viral load maintained at undetectable levels in absence of HAART. He had previously participated in a clinical trial for ustekinumab and reported considerable improvement, so he was restarted on ustekinumab 45 mg every 12 weeks. His HIV viral load prior to initiating therapy was 51 copies/mL and his CD4 count was 997 cells/μL. He experienced rapid relief of his symptoms, with his disease burden improving immensely with near clearance within a few weeks of his first injection. He has now been on ustekinumab for 11 months and has minimal residual disease with no adverse events. At last follow-up, his HIV viral load was 76 copies/mL and his CD4 count was 1,063 cells/μL.

Case 3

Patient 3 is a 66-year-old male with a long-standing history of severe plaque psoriasis and chronic Hepatitis B and HIV infection initially managed on HAART. He has tried and failed several psoriatic treatments, including acitretin, laser therapy, and several topical agents, and was unable to initiate phototherapy because of schedule restraints. Prior to receiving approval from an infectious disease specialist to start biologic therapy, he had not been on psoriasis treatment for two years. On presentation to our clinic, thick scaly plaques covered more than 30% of his body surface area, and he complained of intense pruritus. The patient chose to start adalimumab, however treatment was delayed due to his active Hepatitis B infection. The patient followed with a hepatologist and was started on a concomitant regimen of dolutegravir and lamivudine. Once adalimumab therapy (40 mg every other week) was initiated, the patient experienced near-complete clearing of his skin by week 12. His HIV viral load was less than 40 copies/mL at that time and most recently was undetectable. He is still on adalimumab therapy and has not experienced any adverse events.

Case 4

Patient 4 is a 46-year-old male with a long-standing history of chronic psoriasis, which became much more severe following initial infection with HIV ten years prior to presentation at our clinic. He is an elite controller, with well-controlled HIV viral loads in absence of HAART. He also has a history of foot pain with difficulty walking. After trying and failing several topical agents and phototherapy, he began treatment with methotrexate and experienced complete resolution of his skin and joint symptoms. After five years of methotrexate therapy, he began to experience recurrence of his symptoms. He presented to our clinic at this time to discuss his treatment options. His CD4 count was 350 cells/μL and his HIV viral load was undetectable. The patient was started on etanercept 50 mg twice a week and then weekly after three months. After three months of therapy his plaques nearly

completely resolved, and he has not experienced any adverse events. His most recent CD4 count was 472 cells/μL and his HIV viral load remains undetectable.

Review of biologic therapy in HIV-positive psoriasis patients

Etanercept, Adalimumab, and Infliximab (TNF inhibitors)—To date, 39 cases of HIV-positive patients with psoriasis treated with TNF inhibitors [etanercept (n=31), adalimumab (n=11), or infliximab (n=5)] have been published (including this case series) (Table 1). The majority of these patients report therapeutic success, without serious or opportunistic infections or significant alterations in their CD4 counts or viral loads; however, seven adverse events, including six serious or opportunistic infections, have been reported.

Four cases of HIV-positive psoriasis patients experiencing serious adverse events on etanercept have been reported. In one case, a patient initially experienced dramatic improvement in psoriasis and psoriatic arthritis symptoms on etanercept and HAART, but his treatment course was complicated by frequent polymicrobial infections.[7] In another case, a patient was in the acquired immunodeficiency syndrome (AIDS) stage of disease on etanercept and HAART when he experienced fatal peritonitis, two years after initiating etanercept therapy.[8] Another patient was in the AIDS state of disease on etanercept and HAART when he developed esophageal candidiasis and prostatic adenocarcinoma.[8] Finally, there is a report of a psoriasis patient on etanercept who had undiagnosed HIV disease. Following a year of treatment, the patient experienced significant improvement with a 50% reduction in affected body surface area. However, physical examination at that time also revealed molluscum contagiosum, which resulted in termination of etanercept and patient referral to the HIV clinic.[9]

Three reports of HIV-positive psoriasis patients experiencing serious adverse events on infliximab have been reported. One patient developed miliary TB while on HAART and infliximab, resulting in discontinuation of infliximab despite efficacy in improving psoriasis symptoms.[8] Another patient developed a facial abscess while on HAART and infliximab. The facial abscess responded to antibiotics without complications.[9] Another patient developed a hypersensitivity reaction after his first infliximab injection, but was able to continue therapy by premedicating with corticosteroids. This patient experienced a transitory increase in HIV viral load to 428,503 after his first infliximab injection as well, which was attributed to HAART noncompliance at that time. This did not occur with subsequent injections.[10]

No reports of HIV-positive psoriasis patients experiencing serious adverse events on adalimumab have been reported.

Ustekinumab (IL-12/23 inhibitor), Guselkumab (IL-23 inhibitor)—There have been 16 reported cases of HIV-positive psoriasis patients successfully treated with ustekinumab, an IL-12/23 inhibitor (including this case series) (Table 1). Similar to the TNF inhibitors, reports of ustekinumab in HIV-positive psoriasis patients appear to demonstrate considerable efficacy with reasonable safety profiles. No adverse events have been documented. For the majority of cases, CD4 counts and viral loads actually improved throughout the ustekinumab treatment period. Two patients on ustekinumab experienced a relapse in psoriatic symptoms

at weeks ten and 13, respectively. Starting concomitant phototherapy resulted in increased disease control for one patient.[11] In several patients, including case 1 above, ustekinumab treatment resulted in complete or near-complete clearing of psoriatic lesions following loss of efficacy from etanercept or adalimumab treatment.[2,8,12]

Only one case of a HIV-positive psoriasis patient treated with the IL-23 inhibitor guselkumab has been reported to date. Following six months of treatment, the patient achieved complete clearing of his skin lesions. He did not experience any adverse events, and his viral loads and CD4 counts remained stable throughout treatment.[13]

As of yet, there have been no published reports of HIV-positive patients on other anti-psoriasis biologics secukinumab (IL-17A inhibitor), ixekizumab (IL-17A inhibitor), brodalumab (IL-17RA inhibitor), tildrakizumab (IL-23 p19 inhibitor), risankizumab (IL-23 p19 inhibitor), or certolizumab pegol (TNF inhibitor).

Discussion

This series adds four cases of psoriasis managed by biologic therapy in HIV-positive patients to the 48 that have been published in the dermatologic literature to date (in some cases patients underwent more than one trial of biologic therapy, often due to the lack or loss of efficacy with a particular agent). While data are still limited regarding the efficacy and safety of biologic therapy in the HIV population, available evidence supports the therapeutic efficacy and safety of these agents in treating HIV-positive patients that have moderate-to-severe psoriasis. However, publication bias of successful cases could result in misrepresentation.[13] Randomized controlled trials or even open label trials have not yet been done for HIV patients on biologic therapy, but would help provide objective data on the therapeutic efficacy and safety profiles of these agents in this population.

Due to the often severe, progressive, and refractory nature of psoriasis in HIV-positive patients, biologic therapy is often necessary for disease management in these individuals. For the patients in this case series, several had failed several agents prior to initiating biologic therapy. Due to the U.S. Food and Drug Administration’s warning of potential increased infection risk on psoriasis biologic agents, these drugs may not be routinely prescribed in already immunocompromised HIV patients. This review suggests that with well-controlled HIV disease and regular monitoring of HIV viral loads and CD4 counts, the likelihood of serious or opportunistic infections on these agents may be low. In nearly all cases from the literature, biologic therapy did not negatively impact the CD4 count or HIV viral load, with values often comparable or improving over time.

When choosing a biologic to treat a HIV-positive patient, there are several important considerations. First, the decision should be individualized and involve collaboration with an infectious disease specialist.[14] CD4 counts and HIV viral loads should be assessed pre-treatment to ensure stable HIV status, and regularly assessed throughout treatment to monitor for worsening laboratory values at which point termination should be considered.[14,15]

While there have been more reports of infections in HIV-positive patients on TNF inhibitors versus other biologics, this may be due to the fact that TNF inhibitors have been available for a longer period of time and thus there are more reports of HIV patients on TNF inhibitors.[5] According to 2019 guidelines made by Kaushik and Lebwohl, TNF inhibitors and ustekinumab are all useful in managing psoriasis in HIV-positive patients, as long as their management includes cooperation with an infectious disease specialist and careful monitoring of viral loads and CD4 counts.[14] As for the other psoriasis biologics, data is currently insufficient or unavailable.

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Abbreviations:

HIV	human immunodeficiency virus
HAART	highly active antiretroviral therapy
AIDS	acquired immunodeficiency syndrome

References

1. Mallon E, Bunker CB. HIV-associated psoriasis. AIDS Patient Care STDs. 2000;14(5):239–246. doi:10.1089/108729100317696 [PubMed: 10833810]
2. Paparizos V, Rallis E, Kirsten L, Kyriakis K. Ustekinumab for the treatment of HIV psoriasis. J Dermatol Treat. 2012;23(6):398–399. doi:10.3109/09546634.2011.579085
3. PSORIASIS AND HIV INFECTION - MONTAZERI - 1996 - International Journal of Dermatology - Wiley Online Library. Accessed March 19, 2020. 10.1111/j.1365-4362.1996.tb01658.x
4. Nakamura M, Abrouk M, Farahnik B, Zhu TH, Bhutani T. Psoriasis treatment in HIV-positive patients: a systematic review of systemic immunosuppressive therapies. Cutis. 2018;101(1):38;42;56.
5. Plachouri K-M, Georgiou S Challenges in the treatment of psoriasis with biologics: vaccination, history of malignancy, human immunodeficiency virus (HIV) infection, and pediatric psoriasis. Int J Dermatol. 2019;58(9):1008–1013. doi:10.1111/ijd.14436 [PubMed: 30891751]
6. Gebara NY, El Kamari V, Rizk N. HIV-1 elite controllers: an immunovirological review and clinical perspectives. J Virus Erad. 2019;5(3):163–166. [PubMed: 31700663]
7. Aboulafia DM, Bundow D, Wilske K, Ochs UI. Etanercept for the treatment of human immunodeficiency virus-associated psoriatic arthritis. Mayo Clin Proc. 2000;75(10):1093–1098. doi:10.4065/75.10.1093 [PubMed: 11040859]
8. Montes-Torres A, Aparicio G, Rivera R, et al. Safety and effectiveness of conventional systemic therapy and biological drugs in patients with moderate to severe psoriasis and HIV infection: a retrospective multicenter study. J Dermatol Treat. 2019;30(5):461–465. doi:10.1080/09546634.2018.1535690
9. Kaufman WS, Ahn CS, Huang WW. Molluscum contagiosum in immunocompromised patients: AIDS presenting as molluscum contagiosum in a patient with psoriasis on biologic therapy. Cutis. 2018;101(2):136–140. [PubMed: 29554156]

10. Cepeda EJ, Williams FM, Ishimori ML, Weisman MH, Reveille JD. The use of anti-tumour necrosis factor therapy in HIV-positive individuals with rheumatic disease. Ann Rheum Dis. 2008;67(5):710–712. doi:10.1136/ard.2007.081513 [PubMed: 18079191]
11. Bardazzi F, Magnano M, Campanati A, et al. Biologic Therapies in HIV-infected Patients with Psoriasis: An Italian Experience. Acta Derm Venereol. 2017;97(8):989–990. doi:10.2340/00015555-2698 [PubMed: 28512671]
12. Wieder S, Routt E, Levitt J, Lebwohl M. Treatment of Refractory Psoriasis with Ustekinumab in an HIV-Positive Patient: A Case Presentation and Review of the Biologic Literature. Psoriasis Forum. 2014;20a(3):96–102. doi:10.1177/247553031420a00304
13. Bartos G, Cline A, Beroukhim K, Burrall BA, Feldman SR. Current biological therapies for use in HIV-positive patients with psoriasis: case report of guselkumab used and review. Dermatol Online J. 2018;24(11). Accessed March 20, 2020. https://escholarship.org/uc/item/3db748cg
14. Kaushik SB, Lebwohl MG. Psoriasis: Which therapy for which patient: Focus on special populations and chronic infections. J Am Acad Dermatol. 2019;80(1):43–53. doi:10.1016/j.jaad.2018.06.056 [PubMed: 30017706]
15. Gallitano SM, McDermott L, Brar K, Lowenstein E. Use of tumor necrosis factor (TNF) inhibitors in patients with HIV/AIDS. J Am Acad Dermatol. 2016;74(5):974–980. doi:10.1016/j.jaad.2015.11.043 [PubMed: 26774690]
16. Saeki H, Ito T, Hayashi M, et al. Successful treatment of ustekinumab in a severe psoriasis patient with human immunodeficiency virus infection. J Eur Acad Dermatol Venereol JEADV. 2015;29(8):1653–1655. doi:10.1111/jdv.12531 [PubMed: 24754476]
17. Wang DM, Fernandez AP, Calabrese CM, Calabrese LH. Treatment of psoriasis with ustekinumab in a patient with HIV-related Kaposi sarcoma. Clin Exp Dermatol. 2019;44(1):113–115. doi:10.1111/ced.13630 [PubMed: 29797733]
18. Lindsey SF, Weiss J, Lee ES, Romanelli P. Treatment of severe psoriasis and psoriatic arthritis with adalimumab in an HIV-positive patient. J Drugs Dermatol JDD. 2014;13(7):869–871. [PubMed: 25007373]
19. De Simone C, Perino F, Caldarola G, D'Agostino M, Peris K. Treatment of psoriasis with etanercept in immunocompromised patients: Two case reports. J Int Med Res. 2016;44(1 suppl):67–71. doi:10.1177/0300060515593250
20. Lee ES, Heller MM, Kamangar F, Park KK, Koo JYM. Long-term etanercept use for severe generalized psoriasis in an HIV-infected individual: a case study. J Drugs Dermatol JDD. 2012;11(3):413–414. [PubMed: 22395596]
21. Linardaki G, Katsarou O, Ioannidou P, Karafoulidou A, Boki K. Effective etanercept treatment for psoriatic arthritis complicating concomitant human immunodeficiency virus and hepatitis C virus infection. J Rheumatol. 2007;34(6):1353–1355. [PubMed: 17552060]
22. Lernia VD, Zoboli G, Ficarelli E. Long-term management of HIV/hepatitis C virus associated psoriasis with etanercept. Indian J Dermatol Venereol Leprol. 2013;79(3):444. doi:10.4103/0378-6323.110807
23. Mikhail M, Weinberg JM, Smith BL. Successful Treatment With Etanercept of von Zumbusch Pustular Psoriasis in a Patient With Human Immunodeficiency Virus. Arch Dermatol. 2008;144(4):453–456. doi:10.1001/archderm.144.4.453 [PubMed: 18427038]
24. Sellam J, Bouvard B, Masson C, et al. Use of infliximab to treat psoriatic arthritis in HIV-positive patients. Joint Bone Spine. 2007;74(2):197–200. doi:10.1016/j.jbspin.2006.05.012 [PubMed: 17321777]
25. Bartke U, Venten I, Kreuter A, et al. Human immunodeficiency virus-associated psoriasis and psoriatic arthritis treated with infliximab. Br J Dermatol. 2004;150(4):784–786. doi:10.1111/j.0007-0963.2004.05885.x [PubMed: 15099388]

Table 1.

Four HIV-positive psoriasis patients managed on biologic agents in this case series.

This Case Series											
Patient (sex/age)	**HAART (Y/N)**	**Prior therapies**	**Treatment agents**	**Duration of therapy (months)**	**CD4 count (cells/μL) pre-therapy**	**CD4 count (cells/μL) post-therapy or at last follow-up**	**Viral load (copies/mL) pre-therapy**	**Viral load (copies/mL) post-therapy or at last follow-up**	**Efficacy**	**AEs**	**Other comorbidities**
Case 1 M/48	Y		Ustekinumab Adalimumab, lost efficacy Etanercept, lost efficacy	18 24 36	1,290 1,300 NR	1,652 1,290 1,300	Undetectable Undetectable NA	Undetectable Undetectable Undetectable	Completely clear Well-controlled, then lost efficacy Well-controlled, then lost efficacy	None	None
Case 2 M/58	N, elite controller	Coal tar, prednisone, phototherapy,	Ustekinumab	11	997	1,063	51	76	Nearly clear	None	HBV, alcohol abuse
Case 3 M/66	Y	Topicals, acitretin, laser therapy	Adalimumab	8	NA	NA	< 40	Undetectable	Nearly clear	None	HBV
Case 4 M/46	N, elite controller	Topicals, phototherapy, MTX	Etanercept	6	350	NA	Undetectable	NA	Nearly clear	None	None

Key: Y: yes, N: no, NA: not available, M: male, F: female, AE: adverse event, HAART: highly active antiretroviral therapy, HBV: hepatitis B virus, MTX: methotrexate

Table 2.

HIV-positive psoriasis patients treated with biologics in the dermatologic literature.

HIV-positive psoriasis patients treated with biologics in the dermatologic literature											
Patient (sex/ age)	**HAART (Y/N)**	**Prior therapies**	**Treatment agents**	**Duration of therapy (months)**	**CD4 count (cells/ μL) pre-therapy**	**CD4 count (cells/μL) post-therapy or at last follow-up**	**Viral load (copies/mL) pre-therapy**	**Viral load (copies/mL) post-therapy or at last follow-up**	**Efficacy**	**AEs**	**Other comorbidities**
M/51[13]	Y	Topicals, phototherapy, acitretin, apremilast	Guselkumab	6	Stable	Stable	Stable	Stable	Completely clear	None	Erythrodermic psoriasis
M/61[2]	Y	Etanercept, acitretin, PUVA, CsA, MTX	Ustekinumab	7	429	530	<50	Undetectable	PASI 90 at week 18	None	None
M/53[11]	Y	CsA	Ustekinumab	24	523	454	NA	NA	PASI 75 at week 12	None	HCV, HTN
M/41[11]	Y	CsA	Ustekinumab	35	537	606	NA	NA	PASI 75 at week 12	None	None
M/70[11]	Y	CsA	Ustekinumab	18	186	330	NA	NA	PASI 75 at week 12	None	HTN
F/43[11]	Y	CsA	Ustekinumab	24	535	610	NA	NA	PASI 75 at week 12	None	None
M/39[12]	Y	Phototherapy, hydroxyurea, acitretin, MTX, etanercept, adalimumab, golimumab	Ustekinumab	7	847	856	Undetectable	Undetectable	Significant improvement	NA	PsA
M/47[16]	Y	Etretinate, CsA, NB-UBV	Ustekinumab Adalimumab (lost efficacy)	10 10	755 602	916 755	Undetectable 29	Undetectable Undetectable	Improvement in PASI (92%), psoriasis disability index (25 to 22), and QOL PASI 15.1 » 1.7 (PASI 88) at week 16 (then lost efficacy)	None	NA
M/54[8]	Y	NA	Ustekinumab	10	555	1083	Undetectable	Undetectable	Failed to achieve PASI 75	None	PsA

HIV-positive psoriasis patients treated with biologics in the dermatologic literature											
Patient (sex/ age)	**HAART (Y/N)**	**Prior therapies**	**Treatment agents**	**Duration of therapy (months)**	**CD4 count (cells/ μL) pre-therapy**	**CD4 count (cells/μL) post-therapy or at last follow-up**	**Viral load (copies/mL) pre-therapy**	**Viral load (copies/mL) post-therapy or at last follow-up**	**Efficacy**	**AEs**	**Other comorbidities**
F/35[8]	Y	NA	Ustekinumab	32.5	469	367	4165	26	Failed to achieve PASI 75	None	PsA
M/50[8]	Y	NA	Ustekinumab, Adalimumab, Etanercept	67	722	872	20	Undetectable	PASI 75	None	HCV
M/54[8]	Y	NA	Ustekinumab, Adalimumab, Etanercept	55	350	494	40	Undetectable	Failed to achieve PASI 75	None	HCV
M/46[8]	Y	NA	Ustekinumab, Adalimumab	6	1500	1293	5734845	45	PASI 75	None	NA
M/43[8]	Y	MTX	Ustekinumab, Infliximab	74	876	72	Undetectable	Undetectable	Failed to achieve PASI75	Miliary TB (on infliximab)	NA
M/55[17]	Y	Topicals, phototherapy, acitretin	Ustekinumab	15	212	316	Undetectable	Undetectable	PASI 99	None	Kaposi sarcoma
M/34[11]	N	CsA, PUVA	Adalimumab	26	472	456	NA	NA	PASI 18 » 1.2 (PASI 93)	None	None
M/49[18]	Y	Topical therapy, phototherapy, acitretin	Adalimumab	30	127	550	14649	Undetectable	Complete clearance of skin and near clearance of joint symptoms	None	Low-grade renal cell carcinoma
M/57[11]	Y	CsA, phototherapy	Adalimumab	2	725	800 (still on therapy)	NA	NA	PASI 28 » 5 (PASI 92)	None	HCV, HTN, HLD, carotid stenosis
F/43[8]	Y	NA	Adalimumab, Etanercept	64	1044	2060	80	Undetectable	PASI 75	None	HCV
M/55[11]	Y	CsA, acitretin	Etanercept	72	265	350	NA	NA	PASI 13.4 » 1.2 (PASI 91)	None	HCV, HTN
M/45[11]	Y	CsA, acitretin	Etanercept	16	1,000	1,000	NA	NA	PASI 12 » 0.5 (PASI 96)	None	None
F/38[11]	N	CsA, PUVA, acitretin	Etanercept	2	486	491	NA	NA	PASI 25 » 3.7 (PASI 85)	None	None

HIV-positive psoriasis patients treated with biologics in the dermatologic literature											
Patient (sex/ age)	**HAART (Y/N)**	**Prior therapies**	**Treatment agents**	**Duration of therapy (months)**	**CD4 count (cells/ μL) pre-therapy**	**CD4 count (cells/μL) post-therapy or at last follow-up**	**Viral load (copies/mL) pre-therapy**	**Viral load (copies/mL) post-therapy or at last follow-up**	**Efficacy**	**AEs**	**Other comorbidities**
F/54[11]	Y	CsA	Etanercept	112	870	885	NA	NA	PASI 13.2 » 0.8 (PASI 94)	None	PsA, HCV, HTN
F/33[8]	Y	NA	Etanercept	24	250	380	20	20	PASI 75	None	NA
M/47[8]	Y	NA	Etanercept	68	1132	1.571	Undetectable	Undetectable	PASI 75	None	HBV
M/30[8]	Y	NA	Etanercept	36	400	400	Undetectable	Undetectable	PASI 75	None	PsA, HCV
M/48[8]	Y	NA	Etanercept	24	140	252	240000	750	PASI 75	Fatal acute peritonitis (patient with AIDS)	HCV
M/48[8]	Y	NA	Etanercept	56	671	908	Undetectable	Undetectable	PASI 75	None	HCV
M/55[8]	Y	NA	Etanercept	40	240	NA	20	144	Failed to achieve PASI 75	None	PsA, HCV
M/49[8]	Y	NA	Etanercept	22	423	364	37	37	Failed to achieve PASI 75	None	HCV, HBV
M/48[8]	Y	NA	Etanercept	43	NA	NA	20	20	PASI 75	None	HCV
M/58[8]	Y	NA	Etanercept	34.8	337	880	Undetectable	Undetectable	PASI 75	None	NA
M/64[8]	Y	Efalizumab, CsA	Etanercept	20	NA	394	Undetectable	30.800	NA	None	PsA
M/44[8]	Y	NA	Etanercept	38	NR	755	421	20	NA	None	
M/66[8]	Y	Efalizumab, MTX	Etanercept	109	110	270	149000	143	PASI 75	Esophageal candidiasis, prostatic adenocarcinoma (patient with AIDS)	PsA
M/60[8]	Y	MTX	Etanercept	157	NA	NA	Undetectable	Undetectable	PASI 75	None	PsA
M/50[19]	Y	Topicals, acitretin	Etanercept	6	445	Stable	NA	NA	PASI 24.2 » 1.8 (PASI 93)	None	HBV, HCV
M/56[19]	NA	Topical steroids, acitretin	Etanercept	NA	NA	NA	NA	NA	PASI 26.2 » 3.5 (PASI 87)	None	Alcoholic cirrhosis, liver transplant

HIV-positive psoriasis patients treated with biologics in the dermatologic literature											
Patient (sex/ age)	**HAART (Y/N)**	**Prior therapies**	**Treatment agents**	**Duration of therapy (months)**	**CD4 count (cells/ μL) pre-therapy**	**CD4 count (cells/μL) post-therapy or at last follow-up**	**Viral load (copies/mL) pre-therapy**	**Viral load (copies/mL) post-therapy or at last follow-up**	**Efficacy**	**AEs**	**Other comorbidities**
M/46[20]	Y	Phototherapy, acitretin	Etanercept	78	1370	Stable	Undetected	Undetected	Near complete clearance	NA	NA
M/45[7]	Y	Topicals, phototherapy, steroids, hydroxychloroquine, minocycline, sulfasalazine	Etanercept	6	NA	Stable	NA	Stable	Dramatic improvement in joint & skin inflammation	Frequent polymicrobial infections	PsA
M/43[21]	Y	CsA, MTX	Etanercept	24	380	>450	NA	Undetectable	Nearly clear and PsA remission	None	PsA, HCV, hemophilia
M/51[22]	Y	Phototherapy, prednisolone, acitretin, CsA,	Etanercept	36	200	Stable	7930	Undetectable	Completely clear	None	HCV, erythrodermic psoriasis
M/33[9]	N, underlying HIV not diagnosed at time of treatment	Topicals, MTX	Etanercept	12	NA	60	NA	247,000	BSA from 60 to 30%	Molluscum contagiosum	None
M/32[23]	Y	Topicals	Etanercept	5	NR	Increased to 633	Undetectable	Undetectable	PsA and pustular psoriasis remission by week 4	No infections	PsA, pustular psoriasis
M/28[24]	Y	Topicals, MTX, prednisone	Infliximab	24	425	435	<50	<100	Completely clear and PsA remission	Temporary increase in HIV viral load to 2,818	PsA
M/46[25]	Y	Topicals, acitretin, MTX, phototherapy, prednisone	Infliximab	68	NA	107	NA	Undetectable	Significant improvement in skin and joint symptoms	None	PsA
M/-[24]	Y	MTX, prednisone, acitretin	Infliximab	48	16	233	300,000	5900	Significant improvement in joint & skin symptoms	None	PsA

HIV-positive psoriasis patients treated with biologics in the dermatologic literature											
Patient (sex/ age)	**HAART (Y/N)**	**Prior therapies**	**Treatment agents**	**Duration of therapy (months)**	**CD4 count (cells/ μL) pre-therapy**	**CD4 count (cells/μL) post-therapy or at last follow-up**	**Viral load (copies/mL) pre-therapy**	**Viral load (copies/mL) post-therapy or at last follow-up**	**Efficacy**	**AEs**	**Other comorbidities**
M/39[10]	N	Sulfasalazine, MTX, LEF	Infliximab, Adalimumab, Etanercept	34	750	741	22,148	54,227	Near complete resolution of skin and joint symptoms	Infliximab hypersensitivity reaction, temporary increase in HIV viral load to 428,503	PsA

Key: Y: yes, N: no, NA: not available, M: male, F: female, AE: adverse event, HAART: highly active antiretroviral therapy, PUVA: psoralen plus ultraviolet A, HCV: hepatitis C virus, HBV: hepatitis B virus, HTN: hypertension, HLD: hyperlipidemia, PsA: psoriatic arthritis, CsA: cyclosporine, MTX: methotrexate, AIDS: acute immunodeficiency syndrome, PASI: psoriasis area and severity index