Title
Current biological therapies for use in HIV-positive patients with psoriasis: case report of guselkumab used and review

Journal
Dermatology Online Journal, 24(11)

Authors
Bartos, Gregory
Cline, Abigail
Beroukhim, Kourosh
et al.

Publication Date
2018

DOI
10.5070/D32411041999

Current biological therapies for use in HIV-positive patients with psoriasis: case report of guselkumab used and review

Gregory Bartos[1] BS, Abigail Cline[1] MD PhD, Kourosh Beroukhim[2] MD, Barbara A Burrall[2] MD, and Steven R Feldman[1,3,4] MD PhD

Affiliations: [1]Center for Dermatology Research, Department of Dermatology, Wake Forest School of Medicine, Winston-Salem, North Carolina, USA, [2]Department of Dermatology, University of California Davis School of Medicine, Sacramento, California, USA, [3]Department of Pathology, Wake Forest School of Medicine, Winston-Salem, North Carolina, USA, [4]Department of Social Sciences & Health Policy, Wake Forest School of Medicine, Winston-Salem, North Carolina, USA

Corresponding Author: Abigail Cline MD PhD, Department of Dermatology, Wake Forest School of Medicine, Medical Center Boulevard, Winston-Salem, NC 27157-107, Tel: 336-716-7740, Fax: 336-716-7732, Email: aecline@wakehealth.edu

Abstract

Background: Psoriasis in human immunodeficiency virus (HIV)-positive patients may be severe. Physicians may be tentative to use biologics in HIV-infected patients.

Objective: We present an HIV-positive patient with psoriasis who was treated with guselkumab. This paper aims to investigate the safety, efficacy, and tolerability of biologic therapies for HIV-positive patients with psoriasis.

Methods: A systematic PubMed review of articles dating between 2000-2018 containing key words psoriasis AND HIV, and psoriatic AND HIV combined with several approved biologic therapies. The review generated 15 articles containing 27 cases of HIV-positive patients treated with etanercept, infliximab, adalimumab, or ustekinumab for their psoriasis.

Results: The majority of cases reported excellent clinical responses, limited adverse events, and well tolerated treatment. CD4 count and viral loads were stable throughout treatment. Similar safety and efficacy were seen in the illustrative case report. Available literature is limited to case reports or case series and could be subject to publication bias of successful cases. Many reports lack quantifiable data and report results based on clinical judgement. No randomized, controlled trials evaluate biologic treatment for psoriasis in HIV-positive patients.

Conclusions: The findings suggest that biologic therapy is an efficacious, safe, and tolerable treatment for most patients with moderate-to-severe psoriasis in HIV-positive patients.

Keywords: guselkumab, psoriatic, psoriasis, biologics, etanercept, infliximab, adalimumab, ustekinumab

Introduction

Psoriasis treatment in human immunodeficiency virus (HIV)-positive populations is challenging. Psoriasis can be more severe and refractory to treatment in HIV-positive patients [1]. Specific viral proteins promote interleukin (IL) and tumor necrosis factor (TNF)-alpha expression to aid in viral replication and avoid immune detection [1]. Although, highly active antiretroviral therapy (HAART) is first line treatment for psoriasis in HIV-infected patients, TNF levels are higher in HIV patients with recent initiation of HAART compared to those without therapy [2]. These shared inflammatory cytokines are key mediators in the pathogenesis of psoriasis.

For this reason, biologic therapy that targets these specific cytokines may be required to treat psoriasis in HIV-positive patients (**Figure 1**). Although biologics have revolutionized the treatment of psoriasis, some HIV-positive populations remain untreated. The National Psoriasis Foundation advises cautious use of biologics in HIV-positive patients with moderate-to-severe psoriasis [3]. Anti-TNF and anti-IL therapies warn of increased risk of serious infection compared to non-treated populations [4]. The potential additional hazard in an immunocompromised patient has excluded HIV-

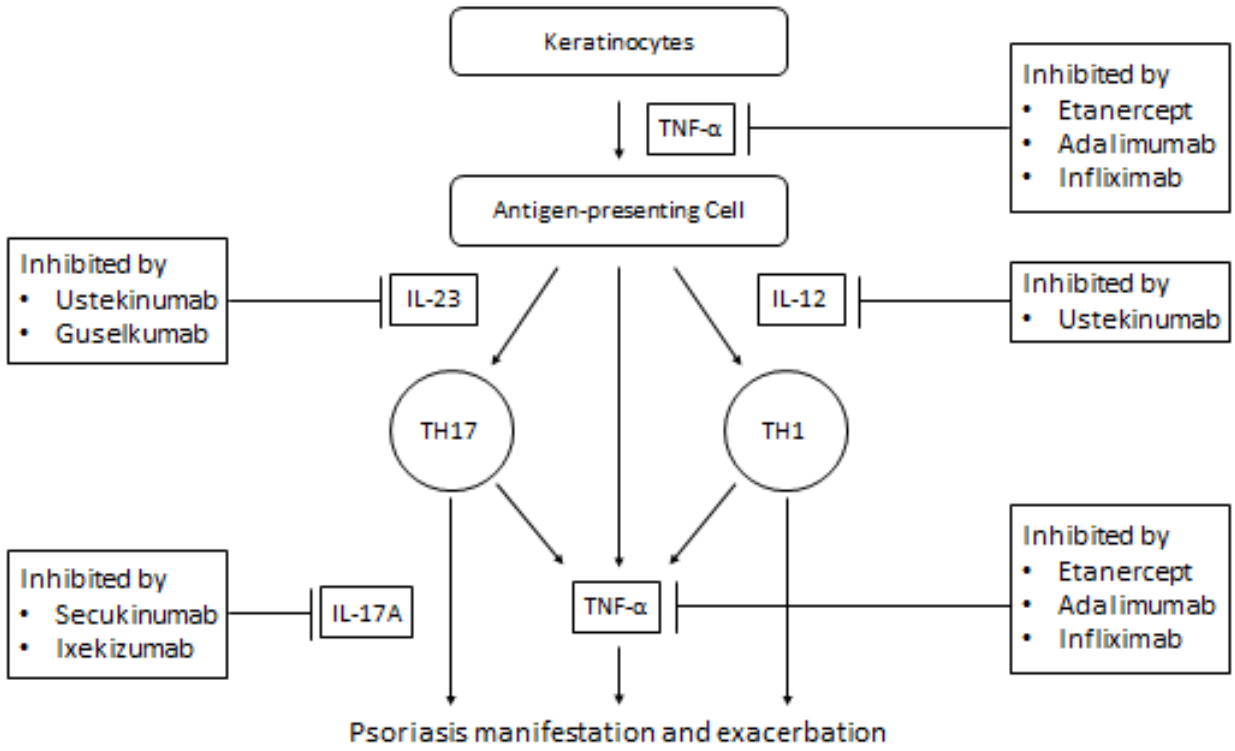

Figure 1. *Schematic representation of biological therapy of psoriasis with the addition of guselkumab [28].*

infected patients from controlled trials, making data regarding efficacy, safety, and tolerability of biologics for psoriasis in HIV-positive patients scarce.

Available literature demonstrating the outcomes of biologic therapy for psoriasis within this challenging population stems from case reports and case series. A recent publication reviewed outcomes of HIV-positive patients receiving systemic therapy for psoriasis [5]. Since its publication, additional cases and trial information were found regarding the use of biologics. This case report and review aims to shed light on the efficacy, safety, and tolerability of biologic therapies for psoriasis within the HIV-positive patients.

Methods

A systematic PubMed review included articles dating between 2000-2018 containing key words psoriasis, HIV, and psoriatic linked with: etanercept, infliximab, adalimumab, ustekinumab, secukinumab, golimumab, tildrakizumab, or certolizumab. Articles earlier than 2000, not in English, or pertaining to pediatrics were omitted. Specific data regarding patient demographics, biologic agent, treatment duration, clinical response, and adverse outcomes were documented. Pre-treatment and post-treatment CD4/viral load levels as well as HAART were also recorded when available.

Our additional illustrative case demonstrates a 51-year-old, HIV-positive male with poorly controlled plaque psoriasis who received guselkumab. Initial management with topical corticosteroids, calcipotriene, and phototherapy was inadequate despite adherence to treatment and HAART. Later, the patient developed erythrodermic psoriasis with concurrent infection and required prolonged hospitalization. Treatment was escalated using oral acitretin and oral apremilast, but only provided temporary benefit. Given his recalcitrant disease covering 30% of his body surface and the desire to minimize additional immunosuppression, the patient was started on guselkumab, 100mg subcutaneously at week 0, week 4, and every 8 weeks thereafter. He has since had complete clearance and has not needed to continue application of topical medications (**Figure 2**). In the intervening six months, CD4 counts remained stable with no significant episodes of abscesses or bacteremia.

Results

Our search yielded 15 articles containing 27 HIV-positive patients who underwent biologic treatment for their psoriasis (**Table 1**). Biologic treatments included etanercept, infliximab, adalimumab, and ustekinumab; no reports contained treatment with golimumab, certolizumab, ixekizumab, or secukinumab. Some patients received more than one biologic owing to poor clinical response [6-8].

Nine reports including 14 patients describe etanercept treatment for HIV-patients. Of these, 93% of patients had partial to excellent clinical responses [6, 8-15]. Twelve of 14 patients were on HAART and showed either an increase or stable CD4 count

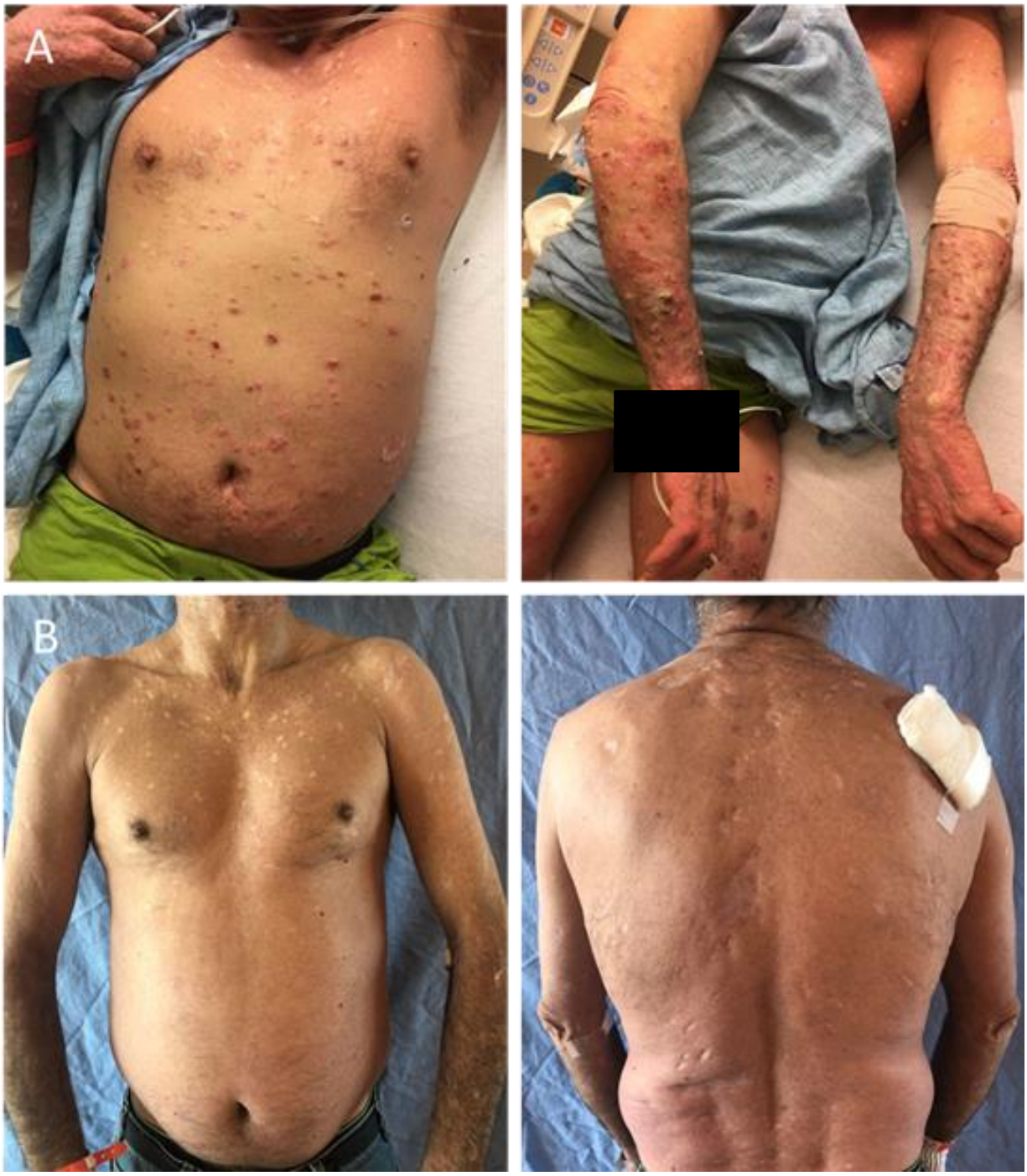

Figure 2. *Treatment of plaque psoriasis with guselkumab in an HIV-positive male. (**A**) The patient was admitted for erythrodermic psoriasis with concurrent infection. (**B**) After 6 months of treatment with guselkumab, the patient had complete clearance of his psoriasis and no significant episodes of abscesses or bacteremia.*

during treatment [6, 8-15]. Adverse events reported in three patients consisted of recurrent infections, allergy, and anemia [6, 8, 14]. Details on the severity and type of anemia were not discussed [8]. Although one patient discontinued therapy after six months owing to recurrent infections, he did have improvement in his psoriasis. Recurrent infections continued after discontinuation and the patient died four months later. This patient was the only one who began treatment with a CD4<50 [14].

Three reports of six cases included infliximab treatment for psoriasis disease with excellent result [6, 16, 17]. All patients on infliximab were on adjunct HAART and displayed no decreases in CD4 count [6, 16, 17]. One case series tested multiple biologics for varying rheumatologic diseases within HIV-positive patients. Three patients with psoriatic arthritis within this series exhibited only partial response to etanercept. Two of the three then switched to adalimumab with comparable results. All three patients then received infliximab and had excellent clinical responses. One adverse event reported was a facial abscess that resolved with antibiotics [6]. One patient had a rise in viral RNA from <50 to 2818 but authors attributed this to poor HAART compliance [16].

Four reports included six HIV-positive patients with psoriasis on adalimumab. Two cases described successful treatment of three patients without adverse events [15, 18]. One patient, not on HAART, had a fall in CD4 count from 472 to 456 [15]. A case series compared the clinical response of etanercept, adalimumab, and infliximab with two patients being placed on adalimumab. This series did not provide detailed information on the adalimumab dosages and duration, but did describe the clinical response as partial without adverse events [6]. Another case described a patient initially started on adalimumab with improvement in PASI score 15.1 to 1.7, but the patient became refractory and PASI worsened from 1.7 to 9.7 despite dosage increase [7].

Five cases reported use of ustekinumab in eight HIV-positive patients with psoriasis. All patients were on HAART with no adverse events [7, 8, 15, 19, 20]. Ustekinumab was successfully used as a second-line biologic in two patients who failed to respond to their initial biologic treatment. These patients had PASI improvements of 92% and 76% after no response to adalimumab and etanercept, respectively [7, 8]. Two patients experienced relapses at 10 and 13 months and required adjunct phototherapy [15]. PASI scores were recorded for seven patients on ustekinumab with an average PASI improvement of 97% [7, 8, 15, 20].

Discussion

Psoriasis in HIV-positive patients appears paradoxical considering the cells and cytokines mediating each process. Historically, psoriasis has been driven by Th1 cytokines associated with CD4+ T-cells whereas HIV is a Th2 process related to CD8+ T-cells [21]. However, the severity of psoriasis paradoxically worsens as CD4+ levels decrease, leading to an immune shift from a Th1 to Th2 predominate cytokines [22]. IL-17, a pro-inflammatory cytokine produced by CD4+ cells, is an important mediator in the pathogenesis of psoriasis and plays an important role in this paradox. Recent assessment of IL-17 secretion found that CD8+ T-cells and NK cells also secrete IL-17 with production potency equal to CD4+ T-cells [23, 24]. HIV-positive patients have a relative increase in CD8+ T-cell levels and CD45RO+ (memory) T-cells, which become major mediators in the pathogenesis of psoriasis by producing inflammatory cytokines like IL-17, IL23, IFN-□, and TNF [21, 25]. These inflammatory cytokines provide targets for biologic therapy and may be helpful to HIV-positive patients with moderate-to-severe psoriasis (**Figure 1**).

This systematic review found 27 reported cases of HIV-positive patients receiving biologic treatment for psoriatic disease (**Table 1**). Biologic agents used were etanercept, infliximab, adalimumab, and ustekinumab. No reports were found for newer biological agents such as secukinumab, ixekizumab, tildrakizumab, golimumab, and certolizumab. Whereas these newer agents have greater efficacy compared to older biologics, there is not as much data regarding their safety and tolerability. However, it may be that the newer, more selective, downstream biologics may prove to be safer (**Figure 1**). This report is the first case of an HIV-positive man

with recalcitrant psoriasis successfully treated with guselkumab.

Physicians may feel more comfortable prescribing biologics that have a decade of data in HIV-positive patients owing to their worry of an adverse event. Reported adverse events include recurrent infection, facial abscess, allergic reaction, infusion reaction, anemia, and rise in viral RNA [6, 8, 14, 16]. The patient who developed a facial abscess while on infliximab had resolution with oral antibiotics [6]. The most severe event reported was a patient on etanercept with a CD4<50 who developed recurrent infections. His CD4 and viral load were stable throughout his treatment. He discontinued etanercept after six months, but continued to have recurrent infections and passed away four months later. Conclusions on the cause of his recurrent infections cannot be made based on treatment resumption after cessation [14]. Our patient displayed none of the reported adverse events and had resolution of his re-occurring abscesses and bacteremia.

CD4 levels and viral load remained stable or improved in 23 of the 27 patients across the four biologics used. Two patients on infliximab had excellent clinical responses but had an increase in viral load. The first patient was not on HAART and his CD4 fell from 750 to 741, while viral load rose from 22,148 to 54,227 [6]. The second patient was on HAART with a stable CD4 count, but his viral load rose from <50 to 2,818 [16]. One patient on ustekinumab and HAART had a fall in CD4 from 523 to 454. In the same case series, a patient on adalimumab without HAART had a decrease in CD4 from 472 to 456 [15]. Overall, stability or improvement of CD4/viral load is likely multifactorial as patients receiving biologic therapy and regular follow-ups were likely more compliant with their HAART [26]. Nevertheless, biologic therapy did not appear to interfere with HAART in maintaining CD4 and viral load. Similar patterns were seen in our patient. Our patient began guselkumab with a CD4 of 54 with a slight decrease to 41 after one year of treatment. Viral load decreased from 29 to undetectable.

Conclusion

Overall, qualitative measurements based on the 15 reviewed articles show predominately positive clinical responses, limited adverse events, tolerance to treatment regimens, and either stable or improvement in CD4 counts and viral load with adjunct HAART. These results warrant future controlled trials without the exclusion of well controlled HIV-positive patients with CD4 counts >200 [27]. Newer biologics such as guselkumab may be safer in HIV or other immunosuppressive states because of their more distal effects. Trials of other biologics in HIV-positive patients are not yet available, but once efficacy, safety, and tolerability are established, biologics may be comfortably extended to HIV-positive patients for moderate to severe, refractory psoriasis.

References

1. Kumar A, Abbas W, Herbein G. TNF and TNF receptor superfamily members in HIV infection: new cellular targets for therapy? *Mediators Inflamm*. 2013;2013:484378. [PMID: 24453421].
2. Wada NI, Jacobson LP, Margolick JB, Breen EC, Macatangay B, Penugonda S, Martinez-Maza O, Bream JH. The effect of HAART-induced HIV suppression on circulating markers of inflammation and immune activation. *AIDS* (London, England). 2015;29(4):463-71. [PMID: 25630041].
3. Menon K, Van Voorhees AS, Bebo BF, Jr., Gladman DD, Hsu S, Kalb RE, Lebwohl MG, Strober BE. Psoriasis in patients with HIV infection: from the medical board of the National Psoriasis Foundation. *J Am Acad Dermatol.* 2010;62(2):291-9. [PMID: 19646777].
4. Singh JA, Wells GA, Christensen R, Tanjong Ghogomu E, Maxwell L, Macdonald JK, Filippini G, Skoetz N, Francis D, Lopes LC, Guyatt GH, Schmitt J, La Mantia L, Weberschock T, Roos JF, Siebert H, Hershan S, Lunn MP, Tugwell P, Buchbinder R. Adverse effects of biologics: a network meta-analysis and Cochrane overview. *The Cochrane database of systematic reviews.* 2011(2):Cd008794. [PMID: 21328309].
5. 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. [PMID: 29529104].
6. 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):710-2. [PMID: 18079191].
7. Saeki H, Ito T, Hayashi M, Fukuchi O, Umezawa Y, Nobeyama Y, Teruya K, Nakagawa H. Successful treatment of ustekinumab in a

severe psoriasis patient with human immunodeficiency virus infection. *J Eur Acad Dermatol Venereol*. 2015;29(8):1653-5. [PMID: 24754476].

8. Paparizos V, Rallis E, Kirsten L, Kyriakis K. Ustekinumab for the treatment of HIV psoriasis. *J Dermatolog Treat*. 2012;23(6):398-9. [PMID: 21787206].
9. 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. [PMID: 27683143].
10. Di Lernia V, 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. [PMID: 23619459].
11. Lee ES, Heller MM, Kamangar F, Park KK, Koo JY. Long-term etanercept use for severe generalized psoriasis in an HIV-infected individual: a case study. *J Drugs Dermatol*. 2012;11(3):413-4. [PMID: 22395596].
12. 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):453-6. [PMID: 18427038].
13. 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. The *J Rheumatol*. 2007;34(6):1353-5. [PMID: 17552060].
14. 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):1093-8. [PMID: 11040859].
15. Bardazzi F, Magnano M, Campanati A, Loconsole F, Carpentieri A, Potenza C, Bernardini N, Di Lernia V, Carrera C, Raone B, Patrizi A, Loi C. Biologic Therapies in HIV-infected Patients with Psoriasis: An Italian Experience. *Acta Derm Venereo*. 2017;97(8):989-90. [PMID: 28512671].
16. Sellam J, Bouvard B, Masson C, Rousiere M, Villoutreix C, Lacombe K, Khanine V, Chennebault JM, Leclech C, Audran M, Berenbaum F. Use of infliximab to treat psoriatic arthritis in HIV-positive patients. *Joint Bone Spine*. 2007;74(2):197-200. [PMID: 17321777].
17. Bartke U, Venten I, Kreuter A, Gubbay S, Altmeyer P, Brockmeyer NH. Human immunodeficiency virus-associated psoriasis and psoriatic arthritis treated with infliximab. *Br J Dermatol*. 2004;150(4):784-6. [PMID: 15099388].
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*. 2014;13(7):869-71. [PMID: 25007373].
19. Wang DM, Fernandez AP, Calabrese CM, Calabrese LH. Treatment of psoriasis with ustekinumab in a patient with HIV-related Kaposi sarcoma. *Clin Ex Dermatol*. 2018. [PMID: 29797733].
20. Wieder S RE, Levitt J, et al. Treatment of refractory psoriasis with ustekinumab in an HIV-positive patient: a case presentation and review of the biologic literature. Psoriasis Forum. 2014(20):96-102. Epub 2018/04/30. DOI: 10.1177/247553031420a00304
21. Fife DJ, Waller JM, Jeffes EW, Koo JY. Unraveling the paradoxes of HIV-associated psoriasis: a review of T-cell subsets and cytokine profiles. *Dermatol Online J*. 2007;13(2):4. [PMID: 17498423].
22. Badger J, Berger TG, Gambla C, Koo JY. HIV and psoriasis. *Clin Rev Allergy Immunol*. 1996;14(4):417-31. [PMID: 9040969].
23. Strzepa A, Szczepanik M. IL-17-expressing cells as a potential therapeutic target for treatment of immunological disorders. *Pharmacol Rep*. 2011;63(1):30-44. [PMID: 21441609].
24. Shin HC, Benbernou N, Fekkar H, Esnault S, Guenounou M. Regulation of IL-17, IFN-gamma and IL-10 in human CD8(+) T cells by cyclic AMP-dependent signal transduction pathway. *Cytokine*. 1998;10(11):841-50. [PMID: 9878122].
25. Duvic M. Immunology of AIDS related to psoriasis. *J Invest Dermatol*. 1990;95(5):38s-40s. [PMID: 2230207].
26. Podsadecki TJ, Vrijens BC, Tousset EP, Rode RA, Hanna GJ. "White coat compliance" limits the reliability of therapeutic drug monitoring in HIV-1-infected patients. *HIV Clin Trials*. 2008;9(4):238-46. [PMID: 18753118].
27. Aukrust P, Liabakk NB, Muller F, Lien E, Espevik T, Froland SS. Serum levels of tumor necrosis factor-alpha (TNF alpha) and soluble TNF receptors in human immunodeficiency virus type 1 infection--correlations to clinical, immunologic, and virologic parameters. *J Infect Dis*. 1994;169(2):420-4. [PMID: 7906293].
28. Dubois Declercq S, Pouliot R. Promising new treatments for psoriasis. *ScientificWorldJournal*. 2013;2013:980419. [PMID: 23935446].

Table 1. *Cases of psoriatic disease in HIV-positive patients treated with biologic therapy.*

Study	Demographics (age/sex)	Biologic Therapy	HAART Yes/No	Treatment Duration (mo)	CD4 Count (cells/mm³)		Viral Load (copies/ml)		Adverse Outcomes	Clinical Response: % Improvement (PASI or BSA)
					Pretreatment	Posttreatment	Pretreatment	Posttreatment		
Bartos, 2018, this study	M/51	Guselkumab	Yes	12	54	41	29	Undetectable	None	Excellent response: 99%
Bardazzi, 2017 [15]	M/53	Ustekinumab	Yes	24	523	454	NA	NA	None	Excellent response: 94%
	M/41	Ustekinumab	Yes	35	537	606	NA	NA	None	Excellent response: 90%
	M/70	Ustekinumab	Yes	18	186	330	NA	NA	None	Excellent response: 93%
	F/43	Ustekinumab	Yes	24	535	610	NA	NA	None	Excellent response: 74%
	M/34	Adalimumab	No	26	472	456	NA	NA	None	Excellent response: 93%
	M/57	Adalimumab	Yes	2	725	800	NA	NA	None	Excellent response: 82%
	F/38	Etanercept	No	2	486	491	NA	NA	None	Excellent response: 85%
	M/45	Etanercept	Yes	16	1000	1000	NA	NA	None	Excellent response: 96%
	M/55	Etanercept	Yes	72	265	350	NA	NA	None	Excellent response: 91%
	F/54	Etanercept	Yes	112	870	885	NA	NA	None	Excellent response: 94%
De Simone, 2016 [9]	50/M	Etanercept	Yes	6	445	Stable	NA	NA	None	Excellent response: 93%
Di Lernia, 2013 [10]	51/M	Etanercept	Yes	36	200-499	Stable	7,930	Undetectable	None	Excellent response: 54%
Lee, 2012 [11]	46/M	Etanercept	Yes	78	1370	>1000	Undetectable	Undetectable	None	Excellent response: NA
Mikhail, 2008 [12]	32/M	Etanercept	Yes	5	435	633	<75	Undetectable	None	Excellent response: 90%
Linardaki, 2007 [13]	43/M	Etanercept	Yes	24	380	>450	<50	Undetectable	None	Excellent response: NA
Aboulafia, 2000 [14]	45/M	Etanercept	Yes	6	<50	Stable	4200	Stable	Frequent infections	Excellent response: NA
Cepeda, 2008 [6]	39/M	Etanercept Infliximab Adalimumab	No	34	750	741	22148	54227	Transient rise in viral RNA, Infusion reaction to Infliximab	Infliximab-Excellent response: NA Etanercept/Adalimumab -Partial response
	52/M	Etanercept Infliximab	Yes	55	268	417	<50	<50	Facial abscess while on Infliximab	Infliximab-Excellent response: NA Etanercept-no response

Table 1, continued. *Cases of psoriatic disease in HIV-positive patients treated with biologic therapy.*

Study	Demographics (age/sex)	Biologic Therapy	HAART Yes/No	Treatment Duration (mo)	CD4 Count (cells/mm^3)		Viral Load (copies/ml)		Adverse Outcomes	Clinical Response: % Improvement (PASI or BSA)
					Pretreatment	Posttreatment	Pretreatment	Posttreatment		
	47/F	Etanercept Infliximab Adalimumab	Yes	13	446	456	<400	<400	Etanercept allergy	Infliximab-Excellent response: NA Etanercept/Adalimumab -Partial response
Sellam, 2007 [16]	28/M	Infliximab	Yes	26	425	435	<50	2818	Rise in viral RNA	Excellent response: NA
	NA/M	Infliximab	Yes	50	16	233	300,000	5900	None	Excellent response: NA
Bartke, 2004 [17]	46/M	Infliximab	Yes	NA	193	107	1040	Undetectable	None	Excellent response: NA
Lindsey, 2014 [18]	49/M	Adalimumab	Yes	30	127	550	14,649	Undetectable	None	Excellent response: 99%
Wang, 2018 [19]	55/M	Ustekinumab	Yes	15	212	316	Undetectable	Undetectable	None	Excellent response: 99%
Saeki, 2015 [7]	47/M	Adalimumab[a] Ustekinumab	Yes Yes	10 NA	602 755	755 916	29 Undetectable	Undetectable Undetectable	None None	Excellent response: 88%[a] Excellent response:92%
Wieder, 2018 [20]	39/M	Ustekinumab	Yes	17	847	856	Undetectable	Undetectable	None	Modest Response: 60%
Paparizos, 2012 [8]	61/M	Etanercept[b] Ustekinumab	Yes Yes	2 14	429 429	Stable 530	Undetectable Undetectable	Undetectable Undetectable	Anemia None	No response[b] Excellent response: 76%

Abbreviations: PASI, Psoriasis Area and Severity Index; BSA, Body Surface Area; HAART, highly active anti-retroviral therapy; M, Male; F, Female; Mo, Months; NA, Not Available.
[a]Patient was initially started on adalimumab with excellent response, but relapsed at month 10, prompting switch to ustekinumab.
[b]Patient was initially started on etanercept with no response, prompting switch to ustekinumab.