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CASE REPORT

Successful Guselkumab Treatment in a Patient with Comorbid Psoriasis and Amyotrophic Lateral Sclerosis: A Case Study and Literature Review

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Abstract: Psoriasis is genetically influenced and can be triggered by factors such as infections, stress, and lifestyle. Chronic plaque psoriasis, the most prevalent form, involves key roles for IL-17 and IL-23 in its pathogenesis. Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder characterized by the degeneration of motor neurons, resulting in muscle weakness and atrophy. Currently, there is no cure for ALS, and treatment is symptomatic, aimed at improving quality of life. The combination of psoriasis and ALS is relatively rare. Although biologic agents have shown remarkable efficacy in the treatment of chronic plaque psoriasis, we have not found any case reports regarding the use of biologic agents for treating psoriasis accompanied by ALS. Our study presents a patient with severe plaque psoriasis and ALS who exhibited a positive response to Guselkumab, without worsening of ALS symptoms, suggesting a promising therapeutic strategy. This could provide a treatment option for patients with psoriasis combined with ALS.We conducted a comprehensive review of the literature on the comorbidity of neurodegenerative diseases, including Alzheimer's disease (AD), Parkinson's disease (PD), multiple sclerosis (MS), and ALS, with plaque psoriasis. This review highlights the differential impact of treatment modalities. Specifically, we found that TNF-α inhibitors may have adverse effects in MS but could provide protective benefits in AD and PD. In ALS patients with psoriasis, IL-17A and IL-23 inhibitors, exemplified by Guselkumab, are suggested as a more suitable alternative due to their lower risk of worsening ALS symptoms.

Keywords: biologics, psoriasis, amyotrophic lateral sclerosis, guselkumab, neurodegenerative disorder

Key Points

- Biologics are widely used in the treatment of moderate to severe plaque psoriasis, but there is limited research on patient with Comorbid Psoriasis and neurodegenerative diseases. Specifically, the safety and efficacy of biologics in psoriasis patients with ALS remain uncertain.
- We reported a case of successful treatment of severe plaque psoriasis combined with ALS using Guselkumab. And literature review found that TNF-α inhibitors may have adverse effects in MS but could provide protective benefits in AD and PD. In ALS patients with psoriasis, IL-17A and IL-23 inhibitors, are suggested as a more suitable alternative due to their lower risk of worsening ALS symptoms.
- Clinicians may consider using IL-23 inhibitors for the treatment of moderate to severe plaque psoriasis in patients with concurrent ALS.

Introduction

Psoriasis is a chronic inflammatory skin disease characterized by erythema, plaques, and scaling, driven by Th1/Th17 cells.[1] This immune-mediated disorder is influenced by genetic and environmental factors and involves cytokines such as TNF-α, IL-17, IL-12, and IL-23.[2,3] Advanced biologic agents and small molecules have emerged as effective, safe treatments for moderate to severe psoriasis, often becoming first-line options.[4] Recognized as a systemic disease,

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psoriasis can have comorbidities such as metabolic syndrome, arthritis, and neuropsychiatric disorders, However, cases of psoriasis comorbid with neurodegenerative diseases (NDs) are rare.[4] ALS is a neurodegenerative disease with a poor prognosis, and cases of psoriasis combined with ALS are even rarer. Currently, there is no specific treatment available, and there is no relevant treatment experience can be learned. Although there is growing interest in biologic treatments for such cases, large-scale studies are scarce. Little is known about the treatment for psoriatic patients with ALS, and we have not retrieved relevant case reports. In order to enrich this clinical vacancy, we report a case of severe plaque psoriasis co-occurring with ALS, successfully treated with Guselkumab, and review the literature on biologic therapies for psoriasis in the context of common NDs, including multiple sclerosis, Alzheimer's, Parkinson's, and ALS.

Case Report

A 72-year-old male with a 20-year history of treatment-resistant plaque psoriasis presented with a recalcitrant disease course. Previous treatments, including phototherapy, acitretin, methotrexate, and various topical agents, had failed to achieve remission. The patient also had a 10-year clinical history of ALS, with a definitive diagnosis made by a neurology team three years ago. His ALS was characterized by progressive motor neuron degeneration, leading to limb weakness, muscle atrophy, and reduced mobility. The coexistence of ALS and psoriasis exacerbated the patient's cutaneous symptoms, which were further worsened by his physical limitations. These limitations hindered his ability to use systemic medications, apply emollients, engage in topical therapies, and perform basic self-care, leading to intractable pruritus and persistent skin lesions. This situation culminated in a significant decline in his health-related quality of life, accompanied by severe depressive episodes and suicidal ideation.

On clinical examination, the patient presented with extensive erythematous plaques and scaling, characteristic of severe psoriasis. His Psoriasis Area and Severity Index (PASI) score was 21.2, with an affected Body Surface Area (BSA) of approximately 38%, and a Dermatology Life Quality Index (DLQI) score of 25, signifying a substantial impact on his daily life. After a comprehensive evaluation for biologic therapy eligibility and exclusion of contraindications, the patient was initiated on Guselkumab, an IL-23 inhibitor, at a dosage of 100 mg via subcutaneous injection at weeks 0, 4, and 12. The therapeutic response was closely monitored with serial PASI assessments. A PASI 50 response, indicative of significant improvement, was achieved by week 4. This improvement progressed to a 60% reduction by week 8 and a 75% reduction by week 12, as illustrated in Figure 1. By the end of the 12-week treatment phase, the patient's PASI score had significantly decreased to 5.2, BSA involvement was reduced to 6.5%, and residual skin lesions were primarily localized to the lower extremities. Concurrently, there was a marked improvement in his DLQI, dropping from 25 to 5, reflecting a substantial enhancement in his dermatological quality of life.

Throughout the treatment period, the patient was under close neurologic surveillance to ensure ALS stability. No exacerbation of ALS symptoms or medication-related adverse effects were observed. This case suggests the potential efficacy of Guselkumab in managing psoriatic symptoms in a patient with ALS, highlighting a promising therapeutic option for this complex comorbidity.

Discussion

In recent years, there has been a expanding body of evidence highlighting the complex interplay between psoriasis and NDs, with a subset of patients demonstrating notable clinical improvements following targeted biologic interventions. While the majority of case reports have centered on the nexus between psoriasis and MS, it is imperative to acknowledge that tumor necrosis factor-alpha (TNF-α) inhibitors, despite their established efficacy in psoriasis management, have been implicated in exacerbating central nervous system (CNS) demyelination and precipitating MS relapses, as well as pontine hemorrhage in rare instances.[5–7] Whereas, the therapeutic targeting of interleukin-17A (IL-17A) and interleukin-23 (IL-23) has gained considerable traction as a viable treatment strategy for psoriasis in the context of MS, offering a more favorable safety profile.[8] However, the application of biologic therapy in psoriasis patients with comorbid chronic NDs, including Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis, remains largely unexplored and necessitates additional research.

Our systematic review endeavors to elucidate the efficacy and safety profiles of biologic agents when employed in the treatment of psoriasis in the backdrop of various common NDs. A comprehensive overview of our findings is presented in Table 1. To date, 11 case reports have detailed the use of biological agents in the treatment of psoriasis among patients with NDs, involving

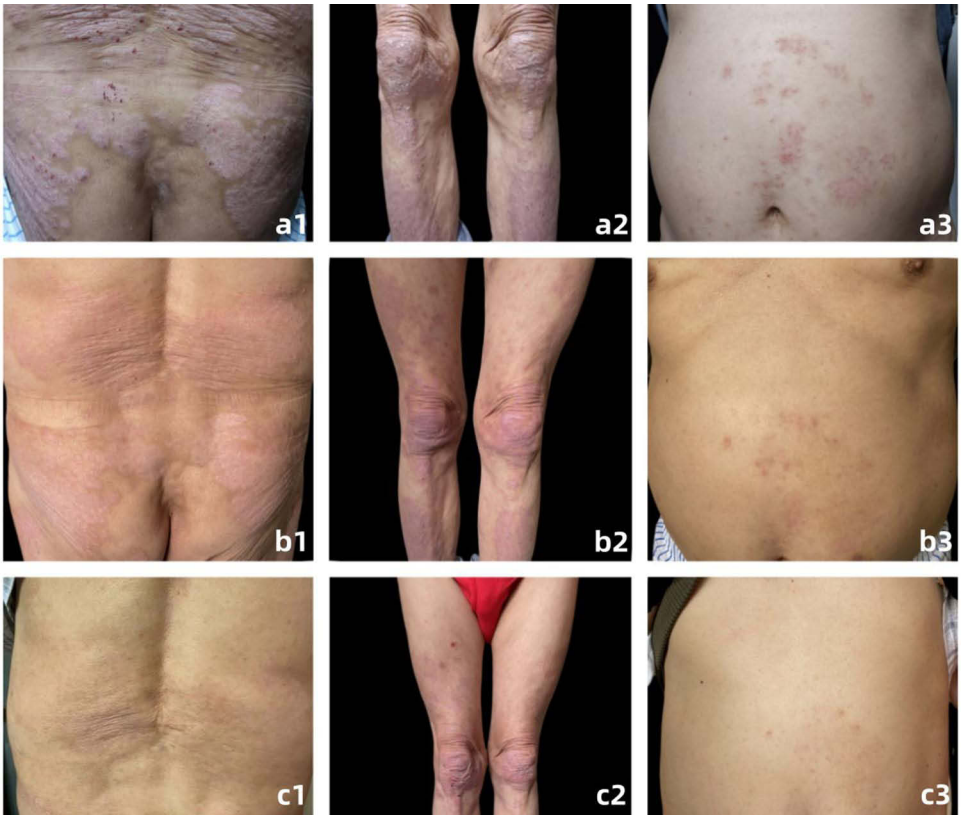

Figure 1 Clinical photographs illustrating the progression of psoriasis in a patient with ALS. (**a**) At baseline, the patient exhibited extensive erythema, plaques, and scaling on the back, lower limbs, and abdomen, with a PASI score of 21.2. (**b**) After 4 weeks of treatment, a noticeable reduction in erythema area and inflammation was observed; PASI score decreased to 8.7. (**c**) By 12 weeks, significant improvement was noted with near-complete resolution of erythema and plaques on the back and abdomen, and residual mild erythema and plaques on the lower limbs, resulting in a PASI score of 5.2.

a total of 12 patients. The majority of case reports have focused on the use of biologics, including secukinumab, ixekizumab, ustekinumab, etanercept, and adalimumab, for treating psoriasis in patients with multiple sclerosis (MS). These reports indicate significant improvements in psoriasis conditions post-treatment. However, MS outcomes vary, with most cases showing stable or worsened conditions, and only one case noting an improvement in MS symptoms. Additionally, one case reported ALS exacerbation following adalimumab treatment for psoriatic arthritis, and another case detailed etanercept treatment for a psoriasis patient with a mental disability, where the mental condition remained stable despite significant skin symptom improvement. The distribution of these cases includes: nine cases of MS treated with secukinumab (SEC, n=5), ustekinumab (UST, n=2), etanercept (ETA, n=1), and ixekizumab (IXE, n=1); one case of amyotrophic lateral sclerosis (ALS) treated with

Table 1 Cases Reported on Biologic Agents for Psoriasis Complicated by Neurodegenerative Diseases

First Author (Year)	Disease	Psoriasis Duration (Years)	Previous Treatments	NDs	Agent	Combined Therapy	Outcomes
Sean A (2006)[13]	PSO	14	UVB,MTX, retinoids	MS	ETA	NA	PASI 100; MS worsening
Elisa (2010)[14]	PSO	11	Photochemotherapy	Mental handicap	ETA	NA	6 months- PASI 100 mental handicap stable
A, Bougea (2014)[15]	PSA	NA	NA	ALS	ADA	NA	PSA stable ALS worsening
Chang Shurong (2015)[16]	PSO & PSA	NA	MTX,efalizumab, acitretin	MS	UST	NA	6 months- PASI 90 MS stable
	PSO & PSA	NA	MTX, IVIG, UVB	MS	UST	NA	4 months- PASI 80 MS stable
Ettler J (2016)[17]	PSO	9	UVB, acitretine, ADA, TA,UST, MTX, CsA, IVIG,corticosteroids	MS	SEC	Prednisone	Psoriasis has significantly improved; MS stable

(*Continued*)

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Table 1 (Continued).

First Author (Year)	Disease	Psoriasis Duration (Years)	Previous Treatments	NDs	Agent	Combined Therapy	Outcomes
Assefa Girum T (2019)[18]	PSO&PSA	23	Tacrolimus, UST	MS	SEC	Prednisolone MTX	2 years- PASI 100, joint pain reduced, stable MS
Diebold Martin (2019)[7]	PSO	6	UVA, UVB, MTX, FAE	MS	SEC	Prednisolone	2 months- PASI 100 Rituximab replaced SEC after MS relapse
M, Venturini (2019)[19]	PSO&PSA	NA	CsA, UVB, ETA, ADA, golimumab, UST, INF, apremilast	MS	SEC	NA	12 weeks- PASI 100 MS stable
Francesca (2020)[20]	PSO&PSA	NA	ETA,INF, ADA, UST, golimumab, certolizumab pegol	MS	SEC	Dimethyl fumarate	3 months- PASI 100, articular improved. MS worsening
Enrique (2021)[21]	PSO	40	MTX, acitretin	MS	IXE	NA	3 years- PASI 0; MS stable
Tsiogkas Sotirios G (2024)[8]	PSO	5	NA	-	IXE	Dimethyl fumarate corticosteroids	4 months- PASI 0 MS appeared after 7 months of treatment with IXE.

Abbreviations: ADA, adalimumab; ALS, Amyotrophic lateral Sclerosis; AD, Alzheimer's Disease; CsA, Cyclosporine A; ETA, etanercept; INF, Infliximab; IXE, ixekizumab; MTX, methotrexate; NA, Not available; PSO, Psoriasis; PSA, Psoriatic Arthritis; MS, Multiple Sclerosis; PD, Parkinson's Disease; PASI, Psoriasis Area Severity Index; SEC, secukinumab; UST, ustekinumab;UVA, Ultraviolet (UV) light, UVA rays; UVB, Ultraviolet (UV) light, UVB rays.

adalimumab (ADA); one case involving a mental handicap treated with EAT; and one case that developed new MS symptoms after 7 months of IXE treatment. It is noteworthy that the literature review identified a dearth of case reports regarding the application of biologics in psoriasis patients with AD or PD. Recent retrospective case-control studies have indicated that TNF-α inhibitors might offer protective benefits against AD development in individuals with chronic inflammatory conditions like psoriasis, ankylosing spondylitis, inflammatory bowel disease, and rheumatoid arthritis.[9] Notably, a groundbreaking 2008 study by Tobinick Edward L et al demonstrated that etanercept, a TNF-α inhibitor, could lead to rapid and substantial improvements in speech in AD patients.[10] Concurrently, emerging evidence suggests that psoriasis may exacerbate the progression of PD, potentially elevating the risk of cognitive decline and depressive symptoms.[11] Furthermore, preliminary data hints at a possible reduction in the risk of PD with the use of tumor necrosis factor-alpha (TNF-α) inhibitors, such as etanercept, adalimumab, and infliximab.[12] However, the mechanisms underlying this potential protective effect are not yet fully understood and remain the subject of ongoing research.

Genome-wide association studies (GWAS) have revealed genetic overlap between NDs and psoriasis. Previous studies have explored potential common pathophysiological mechanisms between psoriasis and conditions such as AD, PD, and MS. For example, in AD, activation of inflammatory cells (primarily microglia) in the brain leads to the release of inflammatory factors that cause neuronal damage.[22] Research has underscored the involvement of the IL-12/IL-23 signaling pathway in amyloid-beta-induced neurodegeneration, a hallmark of AD.[23] Inhibition of the shared p40 subunit of IL-12/IL-23 has demonstrated the potential to reduce amyloid protein production, which may ameliorate clinical symptoms in AD patients. Monoclonal antibodies targeting the IL-12/IL-23 p40 subunit, such as ustekinumab, specifically suppress the IL-23/Th17 axis, a central pathogenic pathway in psoriasis. These antibodies have been recognized as effective treatments for moderate to severe plaque psoriasis worldwide.[24] This mechanistic rationale lends credence to the clinical application of IL-12/IL-23 inhibitors in the management of psoriasis in patients with comorbid AD. However, the therapeutic potential of these agents in this context necessitates further validation through robust clinical trials to ascertain their efficacy and safety profile in treating psoriasis complicated by AD.

Our case represents the inaugural report of Guselkumab utilization in the management of severe plaque psoriasis in the context of ALS. Regarding the ALS condition, we have continued to monitor the patient and observed no worsening of ALS-related symptoms, including progressive weakness, dyspnea, and choking. However, no clinical improvement in ALS symptoms was noted. ALS is a devastating neurodegenerative disorder characterized by the progressive degeneration and loss of motor neurons within the central nervous

system, culminating in muscle paralysis. The etiology of ALS is intricate and multifactorial, with genetic and environmental factors converging to precipitate immune dysregulation as a central pathogenic mechanism.[25] Psoriasis, akin to ALS, originates from a genetic predisposition and is provoked by a spectrum of environmental triggers that incite immune dysfunction. This perturbation involves the IL-23/Th17 axis, which stimulates T helper cells (Th1, Th17) to produce a cascade of cytokines, including IL-23, IL-17A, TNF-α, and IL-12. These cytokines exert their effects on keratinocytes, precipitating abnormal keratinocyte differentiation and hyperkeratosis, which manifest clinically as erythema and plaques.[25] IL-17A, a key cytokine in the pathogenesis of psoriasis, is predominantly produced by Th17 cells. Recent evidence indicates that these cells may contribute to neuroinflammation and potentially worsen neurodegenerative processes. In the context of psoriasis, IL-17A levels within skin lesions and serum are modulated by IL-23, and microglia can augment IL-17A release in the presence of IL-23.[26] Moreover, seminal research conducted by Rentzos et al in 2010, utilizing ELISA assays, revealed that serum and cerebrospinal fluid levels of IL-23 were elevated in ALS patients compared to those with non-inflammatory neurological conditions.[27] The elevated IL-23 levels observed in both ALS and psoriasis suggest that there may be a shared inflammatory pathway. This highlights the potential therapeutic role of IL-23 inhibitors, such as Guselkumab, in treating psoriasis patients with ALS. While the clinical benefit of Guselkumab for psoriasis has been demonstrated, its potential impact on ALS is also worth noting and warrants further investigation.

It's noteworthy that the skin and nervous system, both ectoderm-derived, can be affected by similar diseases, suggesting a common genetic and immunological susceptibility. While Li et al's GWAS found no shared genetic loci between psoriasis and ALS,[28] D'Amico et al identified a correlation between the X-linked SNP rs2294020 in CCDC22 and susceptibility to both conditions.[29] The exact pathogenic overlap between ALS and psoriasis is still under investigation. Some reports link TNF-α inhibitors used for psoriatic arthritis to ALS onset,[15] but no cases have been reported with IL-23 or IL-17A inhibitors. Our case, where Guselkumab successfully treated severe plaque psoriasis in an ALS patient, offers a significant contribution to the literature, providing insights for future clinical strategies for managing such complex cases.

Conclusion

Here, we present a case of severe plaque psoriasis with concomitant ALS, who was successful treated with guselkumab. To our knowledge, this is the first reported case worldwide of a patient with both psoriasis and ALS being treated with Guselkumab. During the 12-week follow-up period, the patient's psoriasis showed significant improvement with resolution of the psoriasis, while the ALS remained stable with no progression of the disease. In our case, Guselkumab has proven to be both effective and safe in treating severe plaque psoriasis in a patient with ALS. This experience suggests that Guselkumab may serve as a promising therapeutic option for managing the complex coexistence of these two conditions. Given the involvement of IL-23 in the pathogenesis of both psoriasis and ALS, we are hopeful that this therapeutic approach might not only improve psoriasis symptoms but also contribute to stabilizing, or even potentially improving, ALS-related manifestations. However, long-term follow-up and additional studies are essential to further confirm its efficacy and safety in this context.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethical Approval and Statement

Ethical approval was not required for this case report as it involves anonymized data and does not include any identifiable patient information. The patient provided informed consent for the publication of their anonymized case details. The patient has given written consent for the publication of their case report and images, with personal identifiers removed to ensure privacy. We confirm that consent was obtained following ethical standards and the patient was fully informed and agreed to the use of their medical information without conditions.

Disclosure

The authors report no conflicts of interest in this work.

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