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Longitudinally Extensive Transverse Myelitis in a Patient with Psoriatic Arthritis: A Therapeutic Dilemma with Clinical Response to Dimethyl Fumarate

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Case Report

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Abstract

Background

Longitudinally extensive transverse myelitis (LETM) is a severe inflammatory myelopathy defined by a T2-hyperintense spinal cord lesion extending across three or more contiguous vertebral segments. Although strongly associated with AQP4-IgG-positive neuromyelitis optica spectrum disorder (NMOSD) and also seen in myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), LETM may occur in other autoimmune settings and, rarely, during immune-modulating therapy. Coexisting PsA creates a therapeutic challenge because neurological treatment may worsen psoriatic disease.

Case presentation:

A 32-year-old man with a 17-year history of PsA, previously controlled with adalimumab, developed progressive paraparesis, a T4/T5 sensory level, paresthesia, constipation, and urinary retention. Approximately a year before presentation, paresthesia and mild left-leg weakness began following an episode of herpes zoster (shingles), with substantial neurological deterioration during the four weeks before referral. Spinal MRI showed a continuous T2–T4 intramedullary lesion. Brain MRI was normal; AQP4-IgG, MOG-IgG, and systemic autoimmune serologies were negative, while CSF showed mild lymphocytic pleocytosis and elevated protein without CSF-restricted oligoclonal bands. Adalimumab was discontinued because of concern for TNF-inhibitor-associated CNS demyelination. After intravenous methylprednisolone, rituximab was started for relapse prevention but was stopped after severe psoriasis involving > 20% of body surface area developed within 12 weeks. Dimethyl fumarate (DMF) was then initiated at 120 mg twice daily for 1 week, followed by 240 mg twice daily. During 2 years of follow-up, neurological function and bladder control improved, psoriasis achieved clinical remission, and MRI demonstrated marked regression of the spinal cord lesion, with no new CNS lesions.

Conclusion

This case highlights the therapeutic complexity of seronegative LETM in a patient with PsA and prior anti-TNF exposure. The temporal association with adalimumab raises, but does not establish, a drug-related mechanism. DMF offered a pragmatic single-agent strategy because of its established activity in demyelinating disorders such as multiple sclerosis, and plaque psoriasis; however, it is not an established maintenance treatment for idiopathic LETM or NMOSD, and this single favorable outcome cannot establish neurological efficacy. The main value of this report lies in the clinical experience it provides and in illustrating the multidisciplinary decision-making required when neuroinflammatory and psoriatic diseases coexist.

Precis

A patient with psoriatic arthritis developed seronegative LETM after long-term adalimumab exposure and experienced severe psoriasis after adalimumab withdrawal and rituximab initiation. DMF was subsequently associated with sustained neurological recovery, MRI improvement, and complete cutaneous remission, highlighting a practical therapeutic option when neurological and psoriatic treatment priorities conflict.

1. Introduction

Psoriasis is a chronic immune-mediated inflammatory disease in which the interleukin-23/-17 (IL-23/IL-17) axis has a central pathogenic role, while psoriatic arthritis (PsA) is its major musculoskeletal manifestation (1). Current PsA recommendations emphasize a domain-based treatment approach that considers peripheral and axial disease, enthesitis, dactylitis, and skin and nail involvement. Biologic and targeted synthetic disease-modifying antirheumatic drugs (DMARDs), including tumor necrosis factor (TNF) inhibitors, are selected according to the patient's clinical phenotype and comorbidities (2).

Longitudinally extensive transverse myelitis (LETM) is defined by spinal cord inflammation extending across at least three contiguous vertebral segments (3). LETM is a recognized neurological phenotype of neuromyelitis optica spectrum disorder (NMOSD) and may also occur in myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), as well as in other inflammatory, infectious, neoplastic, vascular, and drug-associated conditions (4). Testing for aquaporin-4 immunoglobulin G (AQP4-IgG) and myelin oligodendrocyte glycoprotein immunoglobulin G (MOG-IgG) is an important part of the etiological evaluation. However, negative results do not, by themselves, establish an idiopathic diagnosis or exclude an autoimmune myelopathy (3, 5).

The potential association between TNF inhibition and central nervous system (CNS) demyelinating disease remains clinically relevant. Observational studies in patients with spondyloarthritis have reported an association between anti-TNF therapy and neuroinflammatory events, including multiple sclerosis, optic neuritis, and transverse myelitis, although the absolute risk appears to be low (6–8). A 2024 systematic review and meta-analysis involving more than one million patients similarly found an increased pooled risk of inflammatory CNS and demyelinating diseases, but the certainty of evidence was low and residual confounding limited causal inference (9). These findings support vigilance for new neurological symptoms during TNF-inhibitor therapy without establishing causality in individual patients. Management becomes particularly challenging when inflammatory demyelinating disease and active psoriatic disease coexist. Rituximab, although used off-label for NMOSD, can provide effective relapse prevention in selected patients (10). Nevertheless, paradoxical or de novo psoriasis and PsA flares have been reported following B-cell depletion, creating a therapeutic dilemma in patients with concomitant psoriatic disease (11, 12). Thus, treatment directed at controlling CNS inflammation may not necessarily provide adequate control of the associated psoriatic phenotype and may, in some circumstances, be accompanied by worsening of cutaneous or articular manifestations.

Dimethyl fumarate (DMF) is of particular interest in this context because it has established therapeutic roles in both demyelinating events including relapsing multiple sclerosis, and plaque psoriasis (13, 14). Its biological effects extend beyond activation of the nuclear factor erythroid 2-related factor 2 (Nrf2) pathway and include modulation of oxidative stress, inflammatory signaling, cellular metabolism, and neuroimmune responses (14). Current psoriasis treatment guidelines recognize DMF as a systemic treatment option for plaque psoriasis, whereas evidence supporting its use for the articular manifestations of PsA remains limited (15).

Given the rarity and therapeutic complexity of LETM occurring in the setting of PsA, particularly when treatment options are constrained by adverse or paradoxical effects of immunotherapies, clinical experience with alternative therapeutic strategies may be informative. Here, we describe a patient with PsA and LETM who experienced sustained neurological, radiological, and cutaneous improvement following treatment with DMF after treatment-limiting complications associated with anti-TNF therapy and rituximab, highlighting a potential therapeutic strategy in this challenging clinical setting. Figure 1 depicts the timeline of events and findings upon each visit in chronological order (Fig. 1).

2. Case presentation

2. **Case presentation**:
A 32-year-old man with a 17-year history of documented PsA presented with progressive lower-extremity weakness, bilateral paresthesia, constipation, and urinary retention. The patient's PsA began at age 15 and was characterized by symmetric polyarthritis involving the knees, tibiotalar joints, distal interphalangeal (DIP) joints, and proximal interphalangeal (PIP) joints, together with plaque psoriasis of the scalp and extensor surfaces and severe nail pitting. He initially received methotrexate 15 mg/week; persistent joint disease led to adalimumab 10 years before neurological presentation (80 mg loading dose, then 40 mg every 2 weeks), with sustained joint and skin control. Approximately 12 months before referral, the patient experienced herpes zoster (shingles), after which insidious paresthesia and mild left-leg weakness developed. These symptoms initially caused little functional limitation. Four weeks before presentation, weakness rapidly progressed to the right leg, accompanied by severe sensory loss below the chest, constipation, and urinary retention. At presentation, Cranial nerve examination and upper-extremity function were normal. The lower limbs showed severe paraparesis (MRC 2/5 bilaterally), brisk patellar and Achilles reflexes (3+), bilateral Babinski signs, and a T4/T5 sensory level. Whole-spine MRI showed a continuous intramedullary hyperintense lesion from T2 to T4 with subtle enhancement, fulfilling the radiological definition of LETM (Fig. 2). Brain MRI was normal, without lesions suggestive of multiple sclerosis or supportive of a typical NMOSD phenotype. Laboratory tests including AQP4-IgG and MOG-IgG were negative. In addition, ANA, anti-dsDNA, anti-Ro/SSA, anti-La/SSB, ANCA, and antiphospholipid antibodies were also negative. CSF showed mild lymphocytic pleocytosis and elevated protein, with normal glucose and no CSF-restricted oligoclonal bands (OCB). These findings supported an inflammatory myelopathy but did not establish a specific etiology or exclude other infectious, vascular, metabolic, granulomatous, or neoplastic causes. CSF testing for VZV was not performed, limiting assessment of a possible association between the onset of neurological symptoms and herpes

zoster. Because of concern for treatment-associated CNS demyelination, adalimumab was discontinued. Intravenous methylprednisolone 1000 mg/day for 5 days followed by an oral prednisone taper produced moderate neurological stabilization. Rituximab 1000 mg intravenously on days 0 and 14 was then given because of disease severity and concern about recurrence. Within 12 weeks, severe widespread plaque psoriasis involving > 20% of body surface area developed, and rituximab was discontinued. DMF was initiated at 120 mg twice daily for 1 week, followed by an increase to 240 mg twice daily, using a commonly employed multiple sclerosis dosing regimen. During 2 years of follow-up, lower-limb strength improved to 4+/5, sensory deficits resolved, and bladder function normalized. Psoriasis entered complete clinical remission, and MRI showed marked regression and eventual resolution of the T2–T4 lesion without new spinal or brain lesions.

3. Discussion

This case illustrates a therapeutic intersection rather than a new indication for DMF. Severe seronegative LETM, neurological symptoms beginning after herpes zoster, prior anti-TNF exposure, a major psoriasis flare during the transition from adalimumab to rituximab, and subsequent improvement during DMF required simultaneous consideration of neurological and dermatological disease. DMF may be a practical option in selected patients when conventional strategies create competing risks, but its role in inflammatory myelitis remains unproven. The case also supports a broad diagnostic approach to seronegative LETM. Negative AQP4-IgG and MOG-IgG do not identify the cause, and seronegativity does not itself establish idiopathic LETM or exclude all autoimmune myelopathies (16, 17). The temporal association between herpes zoster and the onset of neurological symptoms raises the possibility of VZV-associated spinal cord inflammation. However, the prolonged, progressive course and the absence of definitive virological evidence preclude establishing a causal relationship. A post-infectious inflammatory mechanism is also conceivable but remains speculative. Taken together, these considerations suggest that long-term immunotherapy decisions should account for the severity of the initial attack, the risk of recurrence, diagnostic certainty, and the effects of treatment on coexisting disease. [5]

The dermatological response is consistent with the established role of fumarates in plaque psoriasis, for which DMF remains a systemic treatment option in contemporary guidance (15). This should be distinguished from PsA: evidence for DMF in articular disease is limited, so its role here is primarily as a strategy for cutaneous psoriasis while an immunomodulatory approach to CNS disease is required. Several factors may have contributed to the favorable course. Neurological improvement began after high-dose corticosteroids; adalimumab discontinuation may have removed a potential contributor; and a single seronegative inflammatory myelitis may be monophasic. Thus, the temporal relationship between DMF and sustained neurological recovery is intriguing but does not establish causality. The clinically important observation is the favorable long-term course with simultaneous control of plaque psoriasis after other strategies became problematic. The rationale for DMF was pragmatic and biologically informed rather than based on established evidence for LETM. It addressed the cutaneous component of psoriatic disease while avoiding renewed TNF inhibition and the B-cell depletion associated with the

prior psoriasis flare. Most importantly, DMF is not an established maintenance treatment for idiopathic LETM or double-seronegative NMOSD. Current NMOSD recommendations caution against MS therapies including fumarates in diagnosed NMOSD, particularly AQP4-IgG-positive disease, because of lack of efficacy and reports of severe attacks (18). Nevertheless, since the patients' profile did not fulfill NMOSD criteria, DMF was an attractive choice because it has established clinical activity in plaque psoriasis, and its active metabolite, monomethyl fumarate, modulates neuroimmune response, oxidative stress, inflammatory signaling, cellular metabolism, and immune-cell function, providing a plausible biological framework for activity across inflammatory disorders (14).

Rituximab was followed within 12 weeks by severe plaque-psoriasis exacerbation involving > 20% of body surface area. Although paradoxical psoriasis after B-cell depletion is uncommon, it has been reported, as have PsA flares during rituximab treatment for neuromyelitis optica (11, 12, 19). Proposed mechanisms include altered B-cell–T-cell interactions and Th1/Th17-related immune regulation (19). However, attributing this patient's flare solely to rituximab would overstate the evidence. Adalimumab had provided sustained skin and joint control for years, and its discontinuation preceded the flare; loss of effective suppression or recurrence of the underlying disease is therefore a credible alternative explanation. The close temporal relationship and severity of the flare made continued rituximab treatment clinically unattractive, creating a genuine conflict between neurological disease control and management of psoriatic disease.

Long-term adalimumab exposure adds another consideration. TNF inhibitors are effective for PsA, but rare CNS demyelinating events have been reported. A 2024 meta-analysis involving more than one million patients found an association with inflammatory CNS and demyelinating diseases, although evidence was limited by observational design (9); recent reports include LETM presentations (7, 8). These findings supported discontinuation of adalimumab once the neurological syndrome emerged. However, the approximately 10-year interval between treatment initiation and neurological symptoms makes direct causality uncertain. In addition, herpes zoster occurred before the onset of the neurological syndrome. TNF inhibition can increase susceptibility to herpes zoster at a class level, although drug-specific estimates for adalimumab are inconsistent and some meta-analyses have not found a statistically significant increase for adalimumab alone (20–22). Thus, prior exposure to adalimumab may be relevant to the risk of herpes zoster but does not establish that adalimumab caused either the zoster episode or the subsequent myelopathy. Zoster-associated myelitis is a recognized but rare complication and may present with longitudinally extensive spinal lesions (23, 24). In published cases, neurological symptoms often arise close to the zoster eruption, commonly within days to weeks; a roughly one-year interval in this case is atypical for direct viral invasion. A delayed immune-mediated or post-infectious mechanism is conceivable but remains speculative.

A shared autoimmune susceptibility between psoriasis-spectrum disease and CNS inflammation is biologically plausible, given their complex innate and adaptive immune pathways (25). PsA is strongly associated with IL-23/IL-17-driven inflammation, whereas inflammatory myelopathies involve heterogeneous cellular and humoral mechanisms (1, 3, 26). Whether this patient's LETM was directly

related to PsA, followed zoster through a post-infectious mechanism, or had another cause cannot be determined from the available clinical and laboratory data. The neurological phenotype, continuous T2–T4 T2 hyperintensity, a clear sensory level, severe paraparesis, and bladder dysfunction, is typical of LETM (3). Although LETM is most strongly associated with NMOSD, it also occurs in MOGAD and other inflammatory, infectious, vascular, neoplastic, and systemic autoimmune disorders (27). Negative AQP4-IgG and MOG-IgG results, a normal brain MRI, and the absence of disease-specific systemic autoantibodies did not establish an etiology. The most appropriate diagnosis is seronegative inflammatory LETM of uncertain etiology, with antecedent herpes zoster considered a possible but unproven trigger.

4. Conclusion

This case illustrates a challenging intersection of LETM, PsA, antecedent herpes zoster, and treatment-related uncertainty. Seronegative LETM cannot be confidently attributed to adalimumab or VZV, although the known association between TNF inhibition and rare CNS demyelination justified discontinuation and surveillance. Because neurological symptoms began after zoster but with an approximately one-year interval, VZV-related or post-infectious myelitis remains a hypothesis rather than a confirmed diagnosis. The severe psoriasis flare followed both adalimumab withdrawal and rituximab initiation; loss of prior disease control and a possible rituximab-associated paradoxical flare are competing explanations. DMF was followed by sustained cutaneous control and substantial neurological and radiological recovery over 2 years, but it should be framed primarily as a pragmatic treatment for plaque psoriasis, not an established treatment for LETM or NMOSD. If NMOSD remains a concern, use of DMF warrants particular caution. This single case is hypothesis-generating and supports individualized multidisciplinary care rather than causal or efficacy claims.

Ethical approval and consent to participate:

This study adhered to the ethical standards outlined in the Helsinki Declaration. Written informed consent was obtained from the participant for the publication of this report.

Abbreviations

Psoriatic Arthritis
PsA
Longitudinally Extensive Transverse Myelitis
LETM
Neuromyelitis Optica Spectrum Disorders
NMOSD
Central nervous system
CNS
Magnetic resonance imaging

MRI
Cerebrospinal fluid
CSF
Aquaporin-4
AQP4
Myelin oligodendrocyte glycoprotein
MOG
oligoclonal bands
OCB
Dimethyl Fumarate
DMF
Disease-modifying antirheumatic drugs
DMARDs
Nuclear Factor Erythroid 2-Related Factor 2
Nrf2
Tumor necrosis factor
TNF
Interleukin
IL
T-helper
Th
proximal interphalangeal
PIP
Distal interphalangeal
DIP
Medical Research Council
MRC.

Declarations

Consent for publication:

Written informed consent for publication of the clinical details and images was obtained from the patient. All data have been anonymized to protect patient privacy.

Declaration of interests:

The authors declare that they have no conflicts of interest related to the research, authorship, or publication of this article.

Declaration of Generative AI and AI-Assisted Technologies

ChatGPT (OpenAI) was used during the preparation of this manuscript solely to assist with language editing, including summarizing, grammar correction, improvement of clarity, and refinement of the wording and style of the text. The authors reviewed and revised all AI-assisted content and take full responsibility for the accuracy, originality, and final content of the manuscript.

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Author Contribution

M.E and A.J contributed to study design. M.E and M.H.K performed the clinical study. A.J. and M.H.K. prepared the initial draft of the manuscript. M.E presented the case and details. M.E and A.J amended the manuscript; all authors revised it critically and approved the final version. The authors take full responsibility for the content.

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Data Availability:

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

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Figures

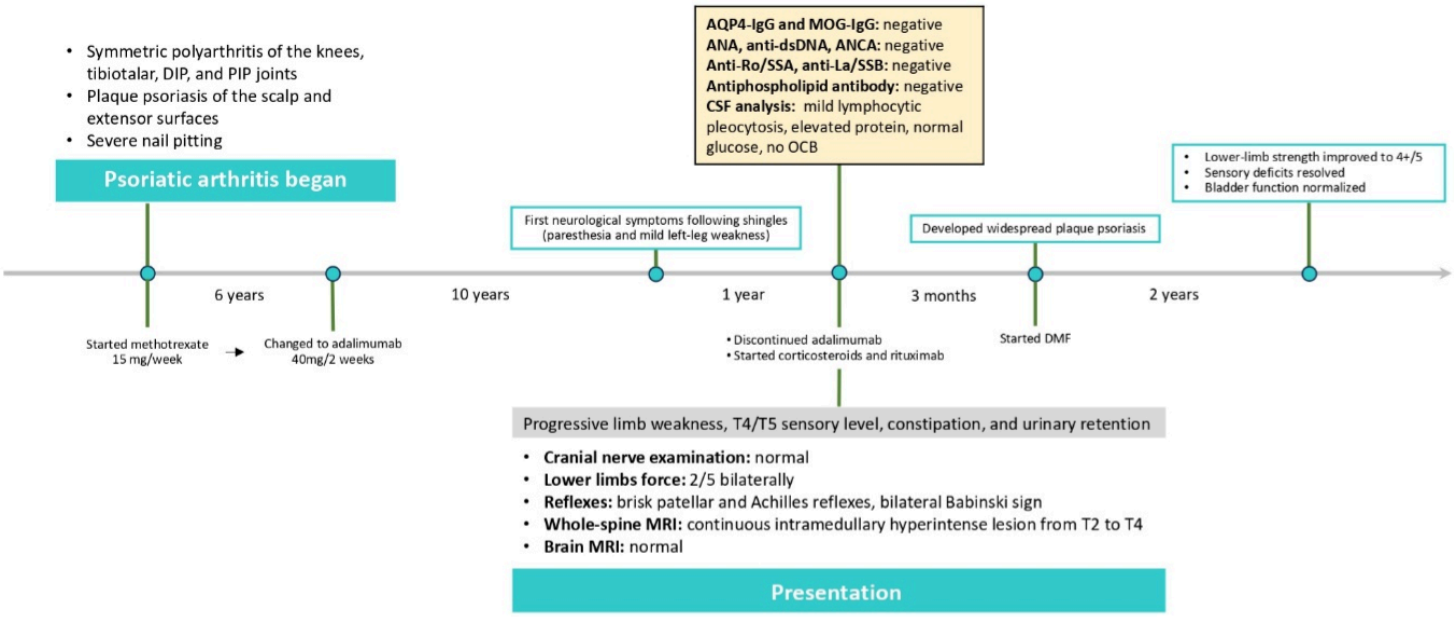


Figure 1

Timeline of the patient's history and visits in chronological order; **abbreviations:** Magnetic resonance imaging, MRI; Cerebrospinal fluid, CSF; Aquaporin-4, AQP4; Myelin oligodendrocyte glycoprotein, MOG; oligoclonal bands, OCB; Dimethyl Fumarate, DMF; proximal interphalangeal, PIP; Distal interphalangeal, DIP

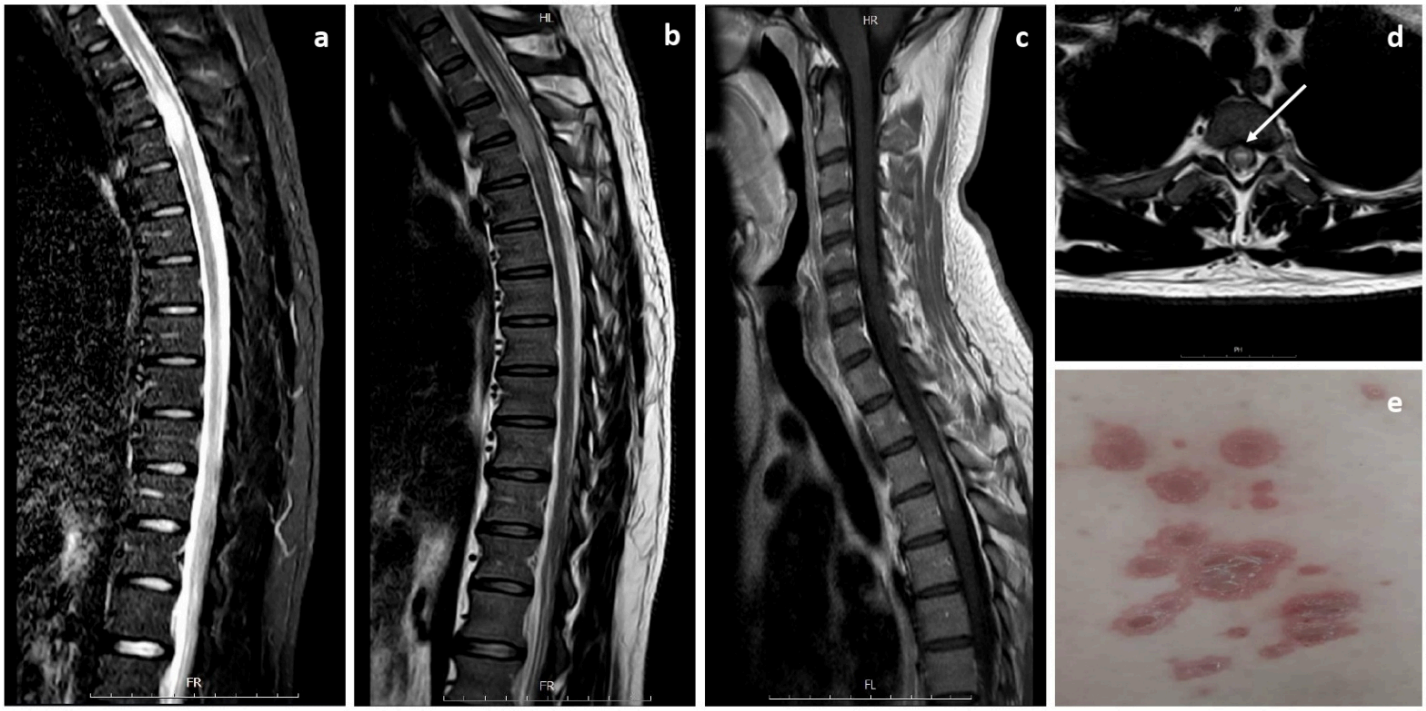


Figure 2

Neurological and dermatological manifestations in a 32-year-old man with longitudinally extensive transverse myelitis and underlying psoriatic arthritis. (a) Sagittal thoracic STIR image showing an intramedullary hyperintense lesion extending from T2 to T4. **(b)** Sagittal T2-weighted image demonstrating the lesion in the same region. **(c)** Sagittal post-gadolinium T1-weighted image showing enhancement within the affected spinal cord segment. **(d)** Axial T2-weighted image at the T2 level demonstrating intramedullary signal abnormality, indicated by the white arrow. **(e)** Cutaneous plaque psoriasis following adalimumab discontinuation and rituximab initiation.

Supplementary Files

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