Drug-Induced Autoimmune Inflammatory Myositis in a Patient with Psoriatic Arthritis Receiving Biologic Therapy: A Case Report

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Abstract

Autoimmune inflammatory myopathies, also referred to as idiopathic inflammatory myopathies, are a heterogeneous group of immune-mediated muscle disorders characterized by muscle inflammation, proximal weakness, and elevated muscle enzymes. Contemporary classification extends beyond the traditional polymyositis–dermatomyositis spectrum to include immune-mediated necrotizing myopathy, antisynthetase syndrome, inclusion body myositis, and overlap myositis, each with distinct clinical and serological features. Drug-induced autoimmune myositis is an increasingly recognized entity, particularly in patients receiving biologic and targeted immunomodulatory therapies, which may disrupt immune homeostasis and trigger inflammatory muscle injury. The authors report the case of a 34-year-old male with psoriatic arthritis on multiple biologic and targeted therapies who presented with acute proximal muscle weakness and markedly elevated creatine phosphokinase levels (2,071 U/L), along with raised inflammatory markers. Vascular causes were excluded on imaging. Based on clinical and biochemical findings, a diagnosis of autoimmune inflammatory myositis was established. The onset of myositis occurred several years after COVID-19 vaccination, making a causal association unlikely. In contrast, the patient's prolonged exposure to immunomodulatory therapies supports a drug-induced etiology. The patient demonstrated clinical improvement following immunosuppressive treatment. This case underscores the importance of recognizing autoimmune inflammatory myositis as a potential complication of biologic therapy and highlights the need for early diagnosis and vigilant monitoring in patients receiving long-term immunomodulation.

Key Points

1. Although uncommon, autoimmune inflammatory myositis is a clinically significant adverse event that has been reported in association with biologic and targeted immunomodulatory therapies used for inflammatory rheumatic diseases, making early recognition essential.
2. This case report describes a patient with psoriatic arthritis who developed autoimmune inflammatory myositis after prolonged sequential exposure to multiple biologic and targeted therapies, highlighting the diagnostic challenge of distinguishing drug-induced myositis from disease progression and other potential causes.
3. In patients receiving long-term biologic or targeted therapy who present with new-onset proximal muscle weakness and markedly elevated creatine phosphokinase levels, clinicians should consider drug-induced autoimmune inflammatory myositis in the differential diagnosis and undertake prompt evaluation and appropriate management.

INTRODUCTION

Autoimmune inflammatory myopathies (AIM), collectively referred to as idiopathic inflammatory myopathies, are a group of heterogeneous immune-mediated disorders characterized by chronic muscle inflammation, progressive weakness, and elevated muscle enzymes.[1-3] Advances in clinical, serological, and histopathological understanding have led to a shift from the traditional classification of polymyositis and dermatomyositis to a more refined categorization that includes dermatomyositis, immune-mediated necrotizing myopathy, antisynthetase syndrome, inclusion body myositis, and overlap myositis.[2-4]

These subtypes are distinguished by specific autoantibodies, clinical features, and patterns of organ involvement, reflecting diverse underlying immunopathogenic mechanisms.[5] The role of environmental triggers, including infections and medications, has been increasingly recognized in the development of autoimmune myositis.[2,6]

Drug-induced autoimmune inflammatory myositis has emerged as an important clinical entity, particularly with the expanding use of biologic agents and targeted synthetic disease-modifying antirheumatic drugs.[6] These therapies, including IL-17 inhibitors and JAK inhibitors, modulate immune pathways but may paradoxically disrupt immune tolerance and precipitate autoimmune phenomena.[2,6]

Psoriatic arthritis is a chronic inflammatory condition frequently managed with such therapies.[7-9] While these treatments have significantly improved disease outcomes, their long-term immunological effects may contribute to rare but clinically significant complications, including inflammatory myopathies.[4,6,7]

In this report, the authors describe a case of autoimmune inflammatory myositis in a patient with psoriatic arthritis receiving multiple biologic therapies, highlighting diagnostic challenges and the importance of recognizing drug-induced etiologies in the modern therapeutic era.

EPIDEMIOLOGY

Autoimmune diseases are a major contributor to increased mortality among middle-aged individuals, with incidence rates varying by condition.[3] Myositis, characterized by muscle inflammation, most commonly occurs between age 45–64 years.[3] Gender differences have been observed, with women more frequently affected by polymyositis and dermatomyositis, and men more commonly diagnosed with inclusion body myositis.[3]

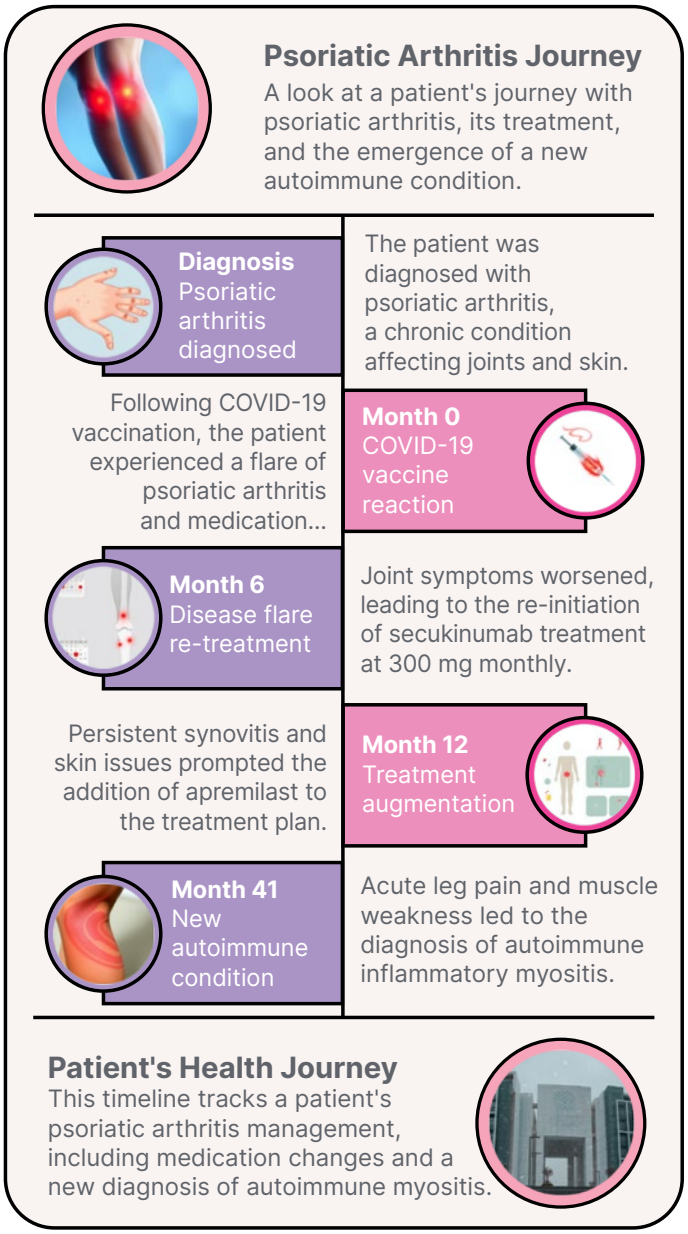
In patients with psoriasis, myositis affects approximately 1.32 per 1,000 individuals.[10] The presence of other autoimmune conditions, psoriatic arthritis, and prior use of anti-TNF-α therapies may elevate the risk of developing myositis in this population.[7-10] Approximately 7,000 new cases of myositis are diagnosed annually in the USA, with a greater incidence observed in certain demographic groups.[3]

CASE PRESENTATION

A 34-year-old man presented to the Department of Rheumatology at a tertiary care hospital in Hyderabad, India, with acute onset of severe pain and swelling in the left lower limb, accompanied by progressive difficulty in walking that eventually required wheelchair assistance. He had a known history of psoriatic arthritis (diagnosed), and had been treated with multiple sequential biologic and targeted immunomodulatory therapies over the course of his disease.

Following his diagnosis, the patient was initiated on secukinumab, which was continued for 4 years. He then received the Covishield™ (Serum Institute of India [SII], Pune, India) COVID-19 vaccine. After experiencing a subsequent flare of his psoriatic arthritis, secukinumab was discontinued, and treatment was switched to tofacitinib. Despite this change, the patient developed worsening joint symptoms and progression of psoriatic skin lesions 6 months later, prompting discontinuation of tofacitinib and re-initiation of secukinumab at a dose of 300 mg monthly. Six months later, recurrent synovitis and worsening cutaneous manifestations were noted, and apremilast was added to his treatment regimen. Owing to persistent disease activity, ixekizumab therapy was initiated 13 months later. The patient subsequently achieved stable clinical improvement throughout the rest of the year and the following year while receiving ixekizumab. However, 1 year and 3 months after ixekizumab therapy

Figure 1: Case timeline.



Clinical timeline of the patient's journey with psoriatic arthritis and autoimmune inflammatory myositis. The figure summarizes the patient's clinical course from the diagnosis of psoriatic arthritis in 2017 through sequential biologic and targeted immunomodulatory therapies, disease flares, treatment modifications, and the onset of autoimmune inflammatory myositis in January 2025, culminating in immunosuppressive treatment and clinical stabilization. The illustration was created by the authors using Piktochart based on the patient's original clinical data.

was initiated, he presented with acute proximal muscle weakness, markedly elevated creatine phosphokinase (CPK) levels, and clinical features suggestive of autoimmune inflammatory myositis. The complete chronology of the patient's treatment course, disease progression, and onset of myositis is illustrated in Figure 1.

The treatment history demonstrated prolonged and sequential exposure to several biologic and targeted immunomodulatory agents over approximately 8 years. Notably, the onset of myositis occurred nearly 4 years after COVID-19 vaccination, whereas the patient had received long-term biologic therapy during the intervening period. This temporal relationship, together with cumulative immunomodulatory exposure, favored a biologic-associated or drug-induced etiology rather than a vaccine-associated mechanism.

On physical examination, the patient was conscious, coherent, and oriented. His vital signs were stable, with a blood pressure of 130/80 mmHg, a heart rate of 88 beats per minute, and an oxygen saturation of 99% on room air. Local examination revealed tenderness, swelling, and increased warmth around the left knee, along with active psoriatic skin lesions. Neuromuscular examination demonstrated symmetrical weakness predominantly involving the proximal muscles of the lower limbs, including the hip flexors, hip extensors, quadriceps, and hamstrings (Medical Research Council [MRC] Grade 3/5), while distal lower limb and upper limb muscle strength remained preserved (Grade 5/5). The findings of manual muscle testing are summarized in Table 1.

The patient did not exhibit clinical features suggestive of specific inflammatory myositis subtypes. In particular, there was no history or examination finding consistent with Raynaud's phenomenon, dysphagia, interstitial lung disease, nail-fold capillary abnormalities, or dermatomyositis-specific

Table 1: Muscle strength assessment (MMT scale).

Muscle group	Side	Strength (MMT Grade)
Hip flexors	Left	3/5
Hip extensors	Left	3/5
Quadriceps	Left	3/5
Hamstrings	Left	3/5
Distal lower limb muscles	Bilateral	5/5
Upper limb muscles	Bilateral	5/5

Interpretation: predominant proximal muscle weakness, consistent with inflammatory myopathy.

MMT: Manual Muscle Testing.

cutaneous manifestations other than the pre-existing psoriatic lesions.

The initial differential diagnosis included cellulitis, deep vein thrombosis, and inflammatory myositis. Laboratory investigations demonstrated markedly elevated serum CPK levels of 2,071 U/L, an elevated C-reactive protein (CRP) level of 60.2 mg/dL, and a mildly increased aspartate aminotransferase (AST) level of 68 U/L. Leukocytosis was also present. A venous Doppler ultrasound examination of both lower limbs revealed no evidence of deep vein thrombosis. Based on the patient's clinical presentation, physical examination findings, and biochemical evidence of muscle injury, a diagnosis of autoimmune inflammatory myositis was established. The laboratory findings are summarized in Table 2.

Further diagnostic evaluation was limited by resource availability. Myositis-specific autoantibody testing, including anti-Jo-1 antibodies, antinuclear antibody (ANA), and extractable nuclear antigen (ENA) panel, was not performed because these investigations were unavailable at the authors' center. In addition, advanced diagnostic modalities, including MRI, electromyography, and muscle biopsy, were not undertaken. The absence of these investigations represents a limitation of this case and precluded further immunological and histopathological characterization of the inflammatory myopathy.

Therapeutic Intervention

Following the diagnosis of autoimmune inflammatory myositis, the patient was treated with immunosuppressive therapy and supportive management aimed at controlling both active muscle inflammation and the underlying psoriatic arthritis.

Methotrexate and leflunomide were administered as immunomodulatory agents to control inflammatory activity, while a short course of tofacitinib was continued as part of the therapeutic strategy. Iguratimod was also prescribed for its anti-inflammatory and immunoregulatory effects. In addition, the patient received symptomatic treatment with tramadol and acetaminophen for pain relief, along with supportive care and close clinical monitoring.

Among these therapies, methotrexate, leflunomide, analgesics, and supportive management were specifically utilized to address the inflammatory myositis and associated muscle symptoms. The treatment approach was individualized considering the coexistence of active psoriatic arthritis and inflammatory muscle involvement.

Outcome and Follow-Up

Following initiation of therapy, the patient demonstrated gradual clinical improvement, including reduction in pain and swelling, improvement in mobility, stabilization of proximal muscle weakness, and overall functional recovery. Inflammatory symptoms improved during follow-up, and the patient was discharged in stable condition with advice for continued immunosuppressive therapy and regular rheumatology follow-up.

Causality Consideration

Although the patient had received COVID-19 vaccination, the onset of myositis occurred 4 years later, indicating no significant temporal relationship. In contrast, the patient had prolonged exposure to multiple biologic and targeted immunomodulatory therapies, supporting a drug-induced etiology.

DISCUSSION

This case highlights the development of AIM in a patient with long-standing psoriatic arthritis receiving multiple sequential biologic and targeted immunomodulatory therapies. Contemporary classification recognizes inflammatory myopathies as a heterogeneous group, including dermatomyositis, immune-mediated necrotizing myopathy, antisynthetase syndrome, overlap myositis, and inclusion body myositis.[2,6,10-11]

Table 2: Laboratory investigation.

Serial number	Parameter	Day 1	Day 2	Reference range
1	Hemoglobin	13.6 g/dL	12.3 g/dL	13.0–17.0 g/dL
2	MPV	10.2 fl	8.2 fl	6.5–12.0 fl
3	WBC	13.04×10³ /mm³	8.76×10³ /mm³	4.0–10.0×10³ /mm³
4	Neutrophils	76.5%	62.8%	40–80%
5	Lymphocytes	12.7%↓	24.3%	20–40%
6	Eosinophils	2.6%	4.4%	1.0–6.0%
7	Monocytes	7.8%	8.0%	2.0–10.0%
8	Basophils	0.4%	0.5%	0–2%
9	Hematocrit	42.3%	39.6%	40–50%
10	MCV	82.2 fl	81.4 fL	83–101 fL
11	MCH	26.4 pg	25.4 pg	27.0-32.0 pg
12	Platelet	344×10³ /mm³	274×10³ /mm³	150–410×10³ /mm³
13	Serum creatinine	0.64 µg/dL	0.71 µg/dL	0.72–1.18 µg/dL
14	CPK	N/A	2,071 U/L↑	39–308 U/L
15	Alkaline phosphatase	87 U/L	N/A	46–116 U/L
16	AST	68 U/L	N/A	<35 U/L
17	ALT	39 U/L	N/A	<45 U/L
18	GGTP	52 U/L	N/A	<55 U/L
19	Sodium (Na+)	142 mmol/L	141 mmol/L	136–145 mmol/L
20	Potassium (K+)	4.1 mmol/L	4.5 mmol/L	3.5–5.5 mmol/L
21	Chloride (Cl-)	105 mmol/L	105 mmol/L	98–107 mmol/L
22	Uric acid	N/A	2.4 mg/dL↓	3.0–8.2 mg/dL
23	C-reactive protein	N/A	60.2 mg/dL↑	0.8–10.0 mg/dL

ALT: alanine transaminase; AST: aspartate aminotransferase; CPK: creatine phosphokinase; GGTP: gamma glutamyl transpeptidase; MCH: mean corpuscular hemoglobin; MCV: mean corpuscular volume; MPV: mean platelet volume; N/A: not applicable; WBC: white blood cells.

A key issue in this case was the previously unclear temporal relationship between COVID-19 vaccination and the onset of myositis. Following revision, a structured clinical timeline demonstrates that myositis developed approximately 4 years after vaccination, making a causal association unlikely. Although rare cases of inflammatory myositis following COVID-19 vaccination have been reported, symptom onset in published cases generally occurs within days to weeks after vaccination. In the present case, myositis developed approximately 4 years after vaccination, making a direct causal relationship unlikely.[12] Therefore, the absence of temporal proximity between vaccination and symptom onset argues against vaccine-induced myositis in this patient.

Instead, the clinical course supports a drug-induced etiology. Drug-induced autoimmune inflammatory myositis associated with biologic and targeted immunomodulatory therapies is increasingly being recognized, although it remains an uncommon clinical entity. Biologic agents are designed to selectively inhibit inflammatory pathways involved in autoimmune diseases; however, paradoxical immune-mediated adverse events have been reported with several classes of biologic therapies.[2,4,5] Proposed mechanisms include cytokine imbalance, altered T cell signaling, loss of immune tolerance, and dysregulated activation of autoreactive immune pathways, ultimately leading to muscle inflammation and injury.[2,5]

In the present case, the patient had prolonged exposure to multiple immunomodulatory agents, including IL-17 inhibitors (secukinumab and ixekizumab) and the JAK inhibitor tofacitinib. These agents modulate key immune pathways and may paradoxically induce autoimmune phenomena through cytokine imbalance, altered T cell signaling, and disruption of immune tolerance.[2,4]

IL-17 inhibitors suppress IL-17-mediated inflammatory signaling, a pathway central to the pathogenesis of psoriasis and psoriatic arthritis. However, IL-17 also plays an important role in maintaining immune homeostasis and host defense. Inhibition of this pathway may result in compensatory immune activation and paradoxical autoimmune manifestations in susceptible individuals.[2] Similarly, JAK inhibitors interfere with intracellular cytokine signaling pathways that regulate immune responses and have been associated with immune dysregulation and paradoxical autoimmune phenomena.[4]

Several reports have described inflammatory myopathies developing following exposure to biologic therapies, particularly TNF-α inhibitors and other targeted biologic agents. Anti-TNF-induced inflammatory myositis has been documented in case series, while recent reports have also described inflammatory myopathies occurring in patients with psoriatic arthritis receiving biologic therapies, supporting the concept of paradoxical immune-mediated adverse events.[4,5,13-15] Although direct causality remains difficult to establish due to the rarity of these reactions and the coexistence of underlying autoimmune disease, the temporal relationship in this patient strongly supports a biologic-associated mechanism. The patient had been receiving sequential biologic and targeted therapies for several years before the onset of myositis, whereas the COVID-19 vaccination had occurred approximately 4 years earlier. Emerging evidence indicates that biologic and targeted therapies may rarely trigger autoimmune inflammatory myopathies through paradoxical immune dysregulation. Several case reports and literature reviews have described inflammatory myositis associated with TNF-α inhibitors, secukinumab, and other biologic therapies, supporting a possible association between prolonged immune modulation and inflammatory muscle disease.[9-15]

Clinically, the diagnosis in this case was supported by proximal muscle weakness and markedly elevated CPK levels. The absence of systemic features such as interstitial lung disease, Raynaud's phenomenon, or dysphagia argues against specific idiopathic inflammatory myositis subtypes and supports a localized inflammatory myopathy. However, the lack of myositis-specific autoantibody testing and advanced investigations such as MRI, electromyography, and muscle biopsy limits precise subtype classification and represents a limitation of this report.[3]

Management of drug-induced AIM involves early recognition, modification or discontinuation of the suspected offending agent, and initiation of immunosuppressive therapy.[4,9] In this patient, treatment with methotrexate, leflunomide, short-course tofacitinib, analgesics, and supportive care resulted in clinical stabilization, reduction in pain, and improvement in functional status, further supporting the proposed diagnosis.

Overall, this case underscores the importance of maintaining a high index of suspicion for inflammatory myopathies in patients receiving biologic and targeted immunomodulatory therapies. With the increasing use of these agents in autoimmune diseases, clinicians should remain aware of rare but clinically significant autoimmune complications, particularly when patients present with unexplained proximal muscle weakness and elevated muscle enzymes.[6]

CONCLUSION

This case highlights the occurrence of autoimmune inflammatory myositis as a potential adverse effect of prolonged biologic and immunomodulatory therapy in a patient with psoriatic arthritis. The absence of temporal association with prior COVID-19 vaccination, along with the patient's treatment history, supports a drug-induced etiology. With the expanding use of biologic and targeted therapies, clinicians should remain vigilant for rare but clinically significant autoimmune complications, including inflammatory myopathies. Early recognition, appropriate evaluation, and timely management are essential to prevent disease progression and improve patient outcomes. This report underscores the importance of careful monitoring and a comprehensive risk–benefit assessment when managing patients on long-term immunosuppressive therapy.

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