

Case report

Not Everything Is Psoriasis

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Running title: Is This Psoriasis?

Abstract

Background: Psoriatic arthritis is a chronic inflammatory musculoskeletal disease associated with psoriasis and characterized by heterogeneous clinical manifestations. Idiopathic inflammatory myopathies and antisynthetase syndrome are distinct autoimmune entities that may rarely coexist with psoriasis or psoriatic arthritis, creating diagnostic and therapeutic challenges. Case presentation: This case report was prepared in accordance with the CARE (CAse REport) reporting guidelines. We present the case of a 42-year-old woman initially diagnosed with psoriatic arthritis after presenting with recurrent knee arthritis accompanied by mild effusion and biopsy-confirmed psoriasis vulgaris localized to the left elbow. The patient fulfilled the CASPAR classification criteria and was initially treated with conventional disease-modifying antirheumatic drugs without satisfactory response. Subsequent treatment with secukinumab also failed

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to achieve disease control. Despite medical recommendations, the patient continued intermittent intramuscular corticosteroid administration prescribed elsewhere for symptomatic relief. Several months later, she was reevaluated because of persistent musculoskeletal complaints and progressive muscle weakness. Physical examination revealed severe proximal muscle weakness, including inability to rise from a prone position. Laboratory investigations demonstrated markedly elevated creatine phosphokinase levels exceeding 3000 U/L. Myositis-specific antibody testing revealed strongly positive anti-Jo-1 antibodies. The diagnosis was revised to inflammatory myopathy compatible with antisynthetase syndrome overlap, and treatment with systemic corticosteroids and azathioprine was initiated. Conclusions: This case highlights the importance of continuous diagnostic reassessment in patients with presumed refractory psoriatic arthritis. Fulfillment of classification criteria should not preclude consideration of additional autoimmune diseases when atypical manifestations emerge. The temporal association with secukinumab also raises the possibility of biologic-related unmasking or triggering of inflammatory myopathy, although causality cannot be established.

Keywords: psoriatic arthritis, anti-Jo-1, inflammatory myopathy, antisynthetase syndrome, secukinumab

1. Introduction

Psoriatic arthritis (PsA) is a chronic immune-mediated inflammatory disease associated with psoriasis and characterized by peripheral arthritis, enthesitis, dactylitis, and axial involvement [1,2]. The CASPAR criteria remain the most widely accepted classification system for PsA and demonstrate high sensitivity and specificity in clinical practice [1].

Dermatomyositis and other idiopathic inflammatory myopathies are rare systemic autoimmune disorders characterized by muscle inflammation, progressive proximal muscle weakness, elevated muscle enzymes, and diverse cutaneous manifestations [3–5]. Psoriasis is a chronic immune-mediated inflammatory disease driven by complex interactions between genetic susceptibility, T-cell activation, and cytokine dysregulation involving the TNF- α , IL-17, and IL-23 pathways [2,6]. Although both diseases share inflammatory immune mechanisms, the coexistence of psoriasis or PsA with inflammatory myopathies remains uncommon and poorly understood [7]. Most currently available evidence derives from isolated case reports and small case series describing overlap syndromes

between psoriasis, dermatomyositis, antisynthetase syndrome, and amyopathic dermatomyositis [7–10].

Idiopathic inflammatory myopathies constitute a distinct group of systemic autoimmune diseases characterized by muscle inflammation, proximal muscle weakness, elevated muscle enzymes, and the presence of myositis-specific antibodies [4,5,11–13]. Among these, antisynthetase syndrome represents a clinically important subset commonly associated with anti-Jo-1 antibodies [14,15], inflammatory myopathy, arthritis, interstitial lung disease, Raynaud phenomenon, fever, and mechanic's hands [16–20].

The coexistence of psoriasis or PsA with inflammatory myopathies is considered uncommon, with only isolated case reports and small case series available in the literature [8,12,15,18]. Such overlap syndromes may create substantial diagnostic uncertainty, particularly when initial manifestations mimic isolated rheumatic disease. Furthermore, biologic therapies used for psoriasis and PsA have occasionally been associated with paradoxical autoimmune phenomena, including inflammatory myopathies [21,22].

We present a case of biopsy-confirmed psoriasis and initially diagnosed PsA in whom progressive proximal muscle weakness and marked hyperCKemia subsequently revealed anti-Jo-1-positive inflammatory myopathy. This case emphasizes the necessity of repeated clinical reassessment and awareness of overlap autoimmune syndromes in patients with refractory disease.

2. Case Presentation

A 42-year-old married woman with two children initially presented with recurrent knee pain accompanied by small joint effusion. Dermatological examination identified a squamous rash localized to the left elbow. Skin biopsy confirmed psoriasis vulgaris.

Based on the presence of inflammatory arthritis and biopsy-proven psoriasis, the patient fulfilled the CASPAR classification criteria for PsA. The patient fulfilled the CASPAR classification criteria with a total score of 4 points, including current biopsy-confirmed psoriasis (2 points), negative rheumatoid factor (1 point), and radiographic evidence of juxta-articular new bone formation (1 point). Initial laboratory evaluation, including creatine phosphokinase (CPK), was within normal reference ranges.

The patient was initially treated with conventional disease-modifying antirheumatic drugs, without significant clinical improvement. Because of

persistent symptoms, treatment with secukinumab was initiated; however, no meaningful therapeutic response was observed.

Despite repeated recommendations to avoid unsupervised corticosteroid administration, the patient continued intermittent intramuscular corticosteroid injections prescribed elsewhere for symptomatic relief of knee pain.

Approximately three months later, the patient was referred again for rheumatologic reevaluation because of persistent musculoskeletal complaints and progressive functional deterioration.

During physical examination, the patient demonstrated severe proximal muscle weakness. Most notably, she was unable to rise from a prone position without assistance, raising suspicion for inflammatory myopathy rather than isolated inflammatory arthritis.

No significant peripheral synovitis beyond the previously described knee involvement was identified at that time.

Repeated laboratory testing demonstrated markedly elevated serum creatine phosphokinase (CPK) levels exceeding 3000 U/L. Further immunological investigation revealed strongly positive anti-Jo-1 antibodies. The patient also reported Raynaud phenomenon. Muscle biopsy demonstrated histopathological findings consistent with idiopathic inflammatory myopathy.

The combination of progressive proximal muscle weakness, severe hyperCKemia, anti-Jo-1 positivity, inflammatory arthritis, Raynaud phenomenon, and muscle biopsy findings supported the diagnosis of anti-Jo-1-positive inflammatory myopathy compatible with antisynthetase syndrome.

Following establishment of the revised diagnosis, treatment with systemic corticosteroids and azathioprine was initiated. The patient demonstrated subsequent clinical improvement, particularly regarding muscle weakness and functional status.

After initiation of immunosuppressive therapy, the patient reported gradual improvement in proximal muscle strength and overall functional capacity. Progressive reduction in muscle-related symptoms was observed during follow-up. No major adverse events related to treatment were documented during the observed follow-up period.

Table 1. Chronological summary of the patient's clinical course.

Time point	Key findings	Management
Initial presentation	Recurrent knee arthritis, biopsy-confirmed psoriasis, normal CPK	Diagnosed with PsA (CASPAR score 4); conventional DMARDs initiated
Persistent disease activity	Ongoing symptoms despite treatment	Secukinumab 300 mg initiated
Six months later	Progressive proximal muscle weakness and Raynaud phenomenon	Diagnostic reassessment performed
Reevaluation	CPK >3000 U/L, anti-Jo-1 positivity, muscle biopsy consistent with inflammatory myopathy	Diagnosis revised to anti-Jo-1-positive inflammatory myopathy compatible with antisynthetase syndrome
Follow-up	Improvement in muscle strength and functional status	Corticosteroids and azathioprine initiated, with subsequent clinical improvement

Abbreviations: CASPAR, Classification Criteria for Psoriatic Arthritis; CPK, creatine phosphokinase; DMARDs, disease-modifying antirheumatic drugs; PsA, psoriatic arthritis.

3. Discussion

PsA and idiopathic inflammatory myopathies are immunologically distinct disorders with different pathogenetic mechanisms, clinical manifestations, and therapeutic strategies. Nevertheless, overlap cases have occasionally been described, presenting substantial diagnostic complexity [8,15,18,19].

Overlap between psoriasis and inflammatory myopathies

The coexistence of psoriasis and inflammatory myopathies represents a rare but increasingly recognized autoimmune overlap phenomenon. Several reports have described associations between psoriasis and dermatomyositis, clinically amyopathic dermatomyositis, juvenile dermatomyositis, and antisynthetase syndrome [8,15,18,19]. Nevertheless, the exact prevalence remains uncertain because most published evidence consists of isolated clinical observations rather than large cohort studies.

The diagnostic process may become particularly challenging because some cutaneous manifestations of dermatomyositis can clinically resemble psoriatic lesions. Gottron papules, erythematous scaling plaques, and inflammatory lesions over extensor surfaces may initially be interpreted as psoriasis, especially in patients with pre-existing psoriatic disease [23,24].

Additionally, inflammatory myopathy-associated arthritis may mimic PsA or other inflammatory arthritides, potentially delaying recognition of systemic muscle involvement [25,26,27].

Several reports have additionally documented vesiculobullous and autoimmune blistering manifestations associated with dermatomyositis [28,29], including bullous pemphigoid, pemphigus foliaceus, dermatitis herpetiformis, linear IgA bullous dermatosis, and vesiculobullous dermatomyositis [30–33]. These observations further support the concept of broad autoimmune overlap and immune dysregulation in inflammatory myopathies [34–36].

Our patient initially fulfilled the CASPAR criteria for PsA because of inflammatory arthritis and biopsy-confirmed psoriasis vulgaris. However, the subsequent development of severe proximal muscle weakness, marked hyperCKemia, and strongly positive anti-Jo-1 antibodies fundamentally altered the diagnostic perspective [37,38,39]. This case therefore illustrates the importance of continuous clinical reassessment in patients with presumed refractory PsA, particularly when atypical manifestations emerge.

This case illustrates a critical principle in rheumatology: classification criteria are not equivalent to definitive diagnosis. Although classification systems are valuable for research standardization and clinical orientation, they should not prevent clinicians from reassessing the diagnosis when atypical manifestations emerge.

One of the most notable aspects of this case was the initially normal CPK level. Normal muscle enzymes at disease onset may create false reassurance and delay recognition of inflammatory myopathy. Repeated evaluation becomes particularly important in patients with refractory symptoms or unexplained functional decline.

An additional factor potentially contributing to diagnostic delay was the patient's repeated intermittent intramuscular corticosteroid administration. Temporary suppression of inflammatory manifestations may have partially obscured the evolution of muscle disease and delayed recognition of progressive myopathy. This possibility should be considered when interpreting the temporal sequence of clinical events in the present case.

Immunopathogenic considerations

The coexistence of psoriasis and inflammatory myopathy may reflect partially overlapping immunologic pathways. Both diseases involve dysregulated activation of innate and adaptive immune responses, including Th1 and Th17-mediated inflammation [2,6]. Cytokines such as TNF- α , IL-17, IL-23, and interferon-associated signaling pathways appear to participate in the pathogenesis of both psoriasis and idiopathic inflammatory myopathies [6,7].

Another possible explanation involves epitope spreading, a phenomenon in which persistent tissue inflammation leads to progressive diversification of autoimmune responses against multiple self-antigens [40]. Such mechanisms may contribute to the emergence of overlap autoimmune syndromes in genetically predisposed individuals.

Secukinumab and possible biologic-related unmasking of myositis

Although causality cannot be definitively established in our patient, biologic therapies have occasionally been implicated in paradoxical autoimmune phenomena. Perna et al. described exacerbation of dermatomyositis following secukinumab therapy in a patient with psoriasis [21]. Similarly, anti-TNF therapy-associated dermatomyositis has also been reported [22].

Our patient had also received secukinumab before the development of overt inflammatory myopathy. Although a direct causal relationship cannot be proven, the temporal association raises the possibility that IL-17 inhibition may have contributed to unmasking or triggering subclinical autoimmune muscle disease in a genetically predisposed individual. Another plausible explanation is that the inflammatory myopathy represented part of the natural evolution of an overlap autoimmune syndrome that became clinically evident despite biologic therapy. Whether biologic therapy directly triggers inflammatory myopathy, unmasks previously subclinical disease, or merely coincides temporally with disease evolution remains uncertain. Nevertheless, these observations emphasize the importance of careful clinical monitoring in patients with refractory inflammatory disease receiving biologic treatment.

Anti-Jo-1 antibodies and diagnostic clarification

Anti-Jo-1 antibodies are among the most common myositis-specific autoantibodies and are strongly associated with antisynthetase syndrome manifestations including inflammatory myopathy, inflammatory arthritis, interstitial lung disease, Raynaud phenomenon, and mechanic's hands [20,40,41]. Autoantibody profiling currently represents a central

component in the classification and prognostic stratification of idiopathic inflammatory myopathies [41,42]. In our patient, the presence of strongly positive anti-Jo-1 antibodies significantly contributed to diagnostic clarification after initial presentation resembling refractory PsA.

Therapeutically, the transition from PsA-directed biologic therapy toward immunosuppressive treatment with corticosteroids and azathioprine resulted in clinical improvement, supporting the inflammatory myopathy diagnosis. This case underscores the necessity of individualized treatment strategies in overlap autoimmune syndromes.

Strengths and Limitations

The strengths of this report include biopsy-confirmed psoriasis, histopathological confirmation of inflammatory myopathy, longitudinal clinical follow-up, and detailed documentation of the diagnostic evolution from presumed psoriatic arthritis to anti-Jo-1-positive inflammatory myopathy compatible with antisynthetase syndrome.

Several limitations should also be acknowledged. This report describes a single patient and therefore cannot establish causality or generalizability. Electromyography and muscle magnetic resonance imaging were not performed. In addition, although inflammatory myopathy became clinically apparent following secukinumab exposure, a causal relationship cannot be established, and the observed temporal association may represent the natural evolution of an underlying autoimmune overlap syndrome.

4. Conclusions

This case demonstrates that persistent or atypical symptoms in presumed PsA should prompt continuous diagnostic reassessment. Fulfillment of classification criteria does not exclude the coexistence of additional autoimmune diseases.

The development of progressive proximal muscle weakness and marked hyperCKemia in our patient ultimately revealed anti-Jo-1-positive inflammatory myopathy overlapping with psoriasis and inflammatory arthritis. Awareness of such rare overlap syndromes is essential for timely diagnosis and appropriate treatment selection.

The temporal relationship between secukinumab exposure and the later recognition of inflammatory myopathy should be interpreted cautiously. It cannot establish causality, but it supports the need for vigilance when new

muscle weakness or unexplained functional decline develops during biologic therapy.

Further studies are needed to better clarify the immunologic relationship between psoriasis, PsA, and inflammatory myopathies, as well as the potential influence of biologic therapies on autoimmune overlap phenomena.

Declarations

Author Contributions

Conceptualization: K.K., Y.B-K.; Methodology: I.Y.; I.S.; Formal analysis: Y.B-K., M.U.; Investigation: I.Y., S.D.; Writing – original draft: K.K., M.U.; Writing – review & editing: M.G-P., S. D. Supervision-M.G. - I.S. Visualisation - G.M-I., V.H. Validation-G.M-I., V.H, S. P-B. Project administration—S.P-B.

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Institutional Review Board Statement

The study was conducted in accordance with the Declaration of Helsinki. Ethical review and approval were not required for this single case report according to local institutional requirements.

Informed Consent Statement

Written informed consent was obtained from the patient for publication of this case report.

Data Availability Statement

The data presented in this study are available on reasonable request from the corresponding author.

Conflicts of Interest

The authors declare no conflict of interest.

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Не всичко е псориазис

Резюме

Въведение: Псориатичният артрит (PsA) е хронично възпалително мускулно-скелетно заболяване, асоциирано с псориазис и характеризиращо се с хетерогенни клинични прояви. Идиопатичните възпалителни миопатии и анти-синтетазният синдром представляват отделни автоимунни заболявания, които рядко могат да съществуват едновременно с псориазис или псориатичен артрит, създавайки диагностични и терапевтични предизвикателства.

Представяне на случая: Представяме случай на 42-годишна жена, първоначално диагностицирана с псориатичен артрит по повод рецидивиращ артрит на колянна става с минимален излив и биопсично потвърден *psoriasis vulgaris*, локализиран по левия лакът. Пациентката покрива CASPAR критериите за PsA и първоначално е лекувана с конвенционални болест-модифициращи антиревматични средства без задоволителен ефект. Последващо лечение със secukinumab също не довежда до контрол на заболяването. Въпреки медицинските препоръки, пациентката продължава периодично приложение на интрамускулни кортикостероиди, назначавани другаде с цел симптоматично облекчаване. Няколко месеца по-късно пациентката е повторно оценена поради персистиращи мускулно-скелетни оплаквания и прогресираща мускулна слабост. Физикалният преглед установява изразена проксимална мускулна слабост, включително невъзможност за изправяне от легнало положение по корем. Лабораторните изследвания показват значително повишени стойности на креатинфосфокиназата над 3000 U/L. Изследването за миозит-специфични антитела установява силно позитивни anti-Jo-1 антитела. Диагнозата е ревизирана към възпалителна миопатия, съвместима с overlap синдром от типа на анти-синтетазен синдром, и е започнато лечение със системни кортикостероиди и azathioprine.

Заключение: Този случай подчертава значението на непрекъснатата диагностична преоценка при пациенти с предполагаем рефрактерен псориатичен артрит. Покриването на класификационните критерии не трябва да изключва възможността за наличие на допълнително автоимунно заболяване при поява на атипични прояви. Времевата връзка със secukinumab повдига и възможността за биологично-индуцирано отключване или демаскиране на възпалителна миопатия, макар причинно-следствена връзка да не може да бъде категорично доказана.

Ключови думи: псориаатичен артрит, анти-синтетазен синдром, anti-Jo-1, възпалителна миопатия, псориазис, secukinumab