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Inflammatory Myopathy Mimicking Polymyalgia Rheumatica: Myalgia With Normal Creatine Kinase and Elevated Aldolase

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABEF 1,2 Hideoki Tanaka
E 1 Keisuke Onoda
E 1 Koji Kawai
E 2 Kazuhiro Koda
EF 2 Hiroaki Koike

1 Department of Internal Medicine, Ito Municipal Hospital, Shizuoka, Japan
2 Department of Internal Medicine, Izu Imaihama Hospital, Shizuoka, Japan

Corresponding Author: Hideoki Tanaka, Department of Internal Medicine, Ito Municipal Hospital, 196-1 Oka, Ito, Shizuoka 414-0055, Japan
Phone: +81-557-37-2626, e-mail: uta.4city@gmail.com

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Patient: Male, 67-year-old
Final Diagnosis: Inflammatory myopathy
Symptoms: Generalized myalgia
Clinical Procedure: —
Specialty: General and Internal Medicine

Objective: Unusual clinical course

Background: Inflammatory myopathies are usually recognized by an elevated creatine kinase (CK) level, and dermatomyositis shows a characteristic rash, but some patients have a normal CK and an elevated aldolase. In an older patient with predominant myalgia and no rash, polymyalgia rheumatica (PMR) is the leading consideration.

Case Report: A 67-year-old man presented with 3 weeks of bilateral shoulder and thigh pain, generalized myalgia, and morning stiffness, with a normal CK level and no rash. He met the PMR classification score threshold at presentation (2012 EULAR/ACR provisional criteria, 6 points); prednisolone 15 mg/day produced only transient relief. Further evaluation revealed elevated aldolase and hyperferritinemia levels, and ground-glass opacities on chest computed tomography. Despite prednisolone 1 mg/kg/day, ferritin rose to 3231 ng/mL and the opacities progressed to consolidation. T2-weighted magnetic resonance imaging showed high signal intensity in proximal muscles. Antibodies against melanoma differentiation-associated gene 5 (MDA5) and aminoacyl-transfer RNA synthetase were detected by enzyme-linked immunosorbent assay, without confirmatory testing. A clinical diagnosis of inflammatory myopathy was made on the combined clinical, biochemical, imaging, and serologic findings. He was transferred for immunosuppressive therapy, and the lung disease stabilized.

Conclusions: An inflammatory myopathy can mimic PMR, presenting with myalgia without a rash or CK elevation. When such a presentation responds inadequately to low-dose glucocorticoids, this possibility should be considered. An elevated aldolase level with a normal CK is a reason to investigate muscle involvement, and myositis-specific autoantibodies are supportive evidence requiring interpretation with the phenotype and assay method. In this patient, the ferritin level increased with radiologic progression and may have reflected disease activity.

Keywords: Creatine Kinase • Ferritins • Aldolase • Myositis • Polymyalgia Rheumatica

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Introduction

Idiopathic inflammatory myopathies are a heterogeneous group of disorders that includes dermatomyositis (DM) and the antisynthetase syndrome. Clinicians recognize these disorders mainly by proximal muscle weakness and elevated creatine kinase (CK), and DM is also recognized by cutaneous findings such as Gottron's papules and heliotrope rash. When neither CK elevation nor a rash is present and myalgia dominates the clinical picture, an inflammatory myopathy is difficult to distinguish from polymyalgia rheumatica (PMR), an inflammatory disorder of older adults that is diagnosed clinically.

Myositis-specific autoantibodies help to define these subtypes. DM associated with antibodies against melanoma differentiation-associated gene 5 (MDA5) usually presents as clinically amyopathic DM, with characteristic cutaneous lesions and little or no muscle involvement [1,2]. It is associated with rapidly progressive interstitial lung disease (ILD), which carries a high early mortality [1-3]. Antibodies against aminoacyl-transfer RNA synthetase (ARS) define the antisynthetase syndrome and are also associated with ILD [2].

Here, we report a case of an older man in whom myalgia predominated and PMR was initially suspected. An elevated aldolase with a normal CK prompted further evaluation, and a clinical diagnosis of inflammatory myopathy with radiologically progressive ILD was subsequently made.

Case Report

The patient was a 67-year-old Japanese man with no relevant medical history who was taking no regular medications. He presented to our hospital with a 3-week history of bilateral shoulder aching, bilateral thigh pain, generalized myalgia, and morning stiffness.

On examination at the initial visit (Day 1), tenderness was present in both thighs and the deltoid regions, and the range of motion of the shoulders and hips was limited by pain. He had no fever, weight loss, or joint swelling, and no respiratory symptoms such as cough or dyspnea. Blood tests showed elevated inflammatory markers; the CK was normal, and rheumatoid factor and anti-cyclic citrullinated peptide (anti-CCP) antibodies were not detected (**Table 1**). With the use of an algorithm that does not incorporate ultrasonography, the patient met the score threshold of the 2012 EULAR/ACR provisional classification criteria for PMR (4 points or more) at presentation; the required criteria of age 50 years or older, bilateral shoulder aching, and an elevated C-reactive protein or erythrocyte sedimentation rate were met, and the score was 6 points (morning stiffness lasting more than 45 minutes, 2 points; limited range of motion of the hips, 1 point; absence of rheumatoid factor and anti-CCP antibodies, 2 points; absence of other joint involvement, 1 point) [4].

On Day 5, treatment with prednisolone (PSL) at a dose of 15 mg/day was started, and the myalgia abated only transiently

Table 1. Laboratory data on Day 1 and on Day 15 (admission).

Variable	Initial visit (Day 1)	On admission (Day 15)	Reference range
Hematology			
White blood cell (/μL)	6565	6970	4000-8000
Red blood cell (× 10^4/μL)	426	456	427-570
Hemoglobin (g/dL)	12.5	13.3	13.5-17.6
Platelet (× 10^4/μL)	45	38.7	15-35
Erythrocyte sedimentation rate (mm/h)	58	83	2-10
Biochemistry			
Total protein (g/dL)	7.0	7.3	6.7-8.3
Albumin (g/dL)	2.7	2.9	3.8-5.3
Total bilirubin (mg/dL)	0.4	0.3	0.2-1.1
Aspartate aminotransferase (U/L)	53	244	10-40
Alanine aminotransferase (U/L)	37	148	5-45
Alkaline phosphatase (U/L)	157	307	43-115
γ-Glutamyl transferase (U/L)	52	270	0-75

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Table 1 continued. Laboratory data on Day 1 and on Day 15 (admission).

Variable	Initial visit (Day 1)	On admission (Day 15)	Reference range
Lactate dehydrogenase (U/L)	360	463	124-222
Creatine kinase (U/L)	96	124	50-250
Aldolase (U/L)	—	17.0	2.7-7.5
Blood urea nitrogen (mg/dL)	13.6	17.5	8-22
Creatinine (mg/dL)	0.73	0.86	0.6-1.1
Sodium (mEq/L)	136	133	135-147
Potassium (mEq/L)	4.2	4.2	3.6-5.0
Chloride (mEq/L)	96	94	98-108
Blood glucose (mg/dL)	183	158	70-110
C-reactive protein (mg/dL)	4.55	2.40	0-0.3
NT-proBNP (pg/mL)	—	107	< 125
KL-6 (U/mL)	—	1220	< 500
Ferritin (ng/mL)	—	1075 (3231*)	21-282
Procalcitonin (ng/mL)	—	0.12	< 0.5
β-D-glucan (pg/mL)	—	5.2	≤ 20
Endocrine			
Thyroid-stimulating hormone (μIU/mL)	—	2.63	0.61-4.23
Free T4 (ng/dL)	—	1.08	0.76-1.65
Immunology			
Soluble interleukin-2 receptor (U/mL)	—	1417	122-496
Antinuclear antibody	Negative	—	Negative
MPO-ANCA	—	Negative	Negative
PR3-ANCA	—	Negative	Negative
Anti-Jo-1 antibody	—	Negative	Negative
Anti-MDA5 antibody (index)	—	770**	< 32
Anti-ARS antibody (index)	—	58.3**	< 25

* Peak ferritin level during hospitalization (Day 18); ** Measured by enzyme-linked immunosorbent assay (ELISA) at an external laboratory. Em dashes (—) indicate that the test was not performed on that day. ANCA, antineutrophil cytoplasmic antibody; ARS, aminoacyl-tRNA synthetase; KL-6, Krebs von den Lungen-6; MDA5, melanoma differentiation-associated gene 5; MPO, myeloperoxidase; NT-proBNP, N-terminal pro-brain natriuretic peptide; PR3, proteinase 3.

and incompletely. Because of worsening malaise, the patient was admitted to our hospital on Day 15; he had no respiratory symptoms at that time. On examination, tenderness in the thighs and deltoid regions persisted, and manual muscle testing showed mild proximal weakness (4/5), with normal distal strength (5/5). Pain was present during testing; the examining physician judged the reduced proximal strength to reflect true weakness, although a contribution of pain-limited effort to the result cannot be excluded. Examination of the skin revealed no heliotrope rash, Gottron's papules, or mechanic's hands. Laboratory testing showed an elevated aldolase (17.0 U/L; reference range, 2.7 to 7.5) with a normal CK (124 U/L). Aspartate aminotransferase was 244 U/L, alanine aminotransferase 148 U/L, and gamma-glutamyl transferase

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(GGT) 270 U/L. Ferritin was elevated at 1075 ng/mL, KL-6 at 1220 U/mL, and the soluble interleukin-2 receptor was also elevated (**Table 1**). Procalcitonin and beta-D-glucan were normal, and tests for infectious and alternative causes, including an interferon-gamma release assay and assays for hepatitis B virus, Epstein-Barr virus, cytomegalovirus, and parvovirus B19, were negative. Computed tomography (CT) of the chest on admission revealed faint ground-glass opacities in the dorsal portions of both lower lobes (**Figure 1A, 1B**).

Because of the inadequate response to low-dose PSL and the clinical suspicion of an underlying inflammatory myopathy, the PSL dose was increased to 50 mg/day (1 mg/kg/day), and the myalgia resolved. By Day 18, however, ferritin had risen to 3231 ng/mL, and CT of the chest on Day 19 showed progression of the ground-glass opacities with new consolidation (**Figure 1C, 1D**). Throughout this period, the oxygen saturation remained at 95% while the patient was breathing ambient air. On Day 22, T2-weighted magnetic resonance imaging (MRI) of the pelvis and thighs, obtained without fat suppression, showed high signal intensity in the gluteus maximus and vastus intermedius muscles on both sides, with a right-sided predominance (**Figure 1E, 1F**).

Testing for myositis-specific autoantibodies (MSAs) by enzyme-linked immunosorbent assay (ELISA) was positive for antibodies against both MDA5 and ARS. Anti-Jo-1 antibodies were not detected. Antinuclear antibody testing was negative, including for cytoplasmic staining patterns. These results were interpreted together with the myalgia, mild proximal muscle weakness, an elevated aldolase, the MRI findings, and progressive ILD, and a clinical diagnosis of inflammatory myopathy was made. Because of the risk of respiratory failure, the patient was transferred to a tertiary care center on Day 23.

At the tertiary care center, treatment was started without additional procedures such as muscle biopsy. No rash developed at any point during the clinical course, including at that center. The patient received multimodal immunosuppressive therapy consisting of intravenous methylprednisolone pulse therapy, tacrolimus, cyclophosphamide, plasma exchange, and a Janus kinase inhibitor, and the ILD stabilized. The referral records described this regimen only in summary form, and the specific Janus kinase inhibitor and the doses were not available. He was discharged after a 2-month hospitalization and has remained under outpatient follow-up for 5 months.

Discussion

The main lesson from this case is that an inflammatory myopathy can closely resemble PMR when myalgia predominates and neither a rash nor CK elevation is present. At presentation, the patient met the score threshold of the 2012 EULAR/ACR provisional classification criteria for PMR [4], and the initial presentation was consistent with PMR. The inadequate response to low-dose PSL prompted us to broaden the differential diagnosis, although such a response does not itself exclude PMR [5].

The diagnostic label in this case requires qualification. Under the 2017 EULAR/ACR classification criteria for idiopathic inflammatory myopathies, the version without muscle biopsy gave a score of 5.8 points (age at onset of 40 years or older, 2.1; symmetric proximal weakness of the upper extremities, 0.7, and of the lower extremities, 0.8; proximal leg muscles weaker than distal leg muscles, 0.9; elevated serum lactate dehydrogenase and aminotransferases, 1.3), which meets the threshold of 5.5 points for probable idiopathic inflammatory myopathy [6]. Because pain may have influenced the strength testing and the enzyme elevations may have had a hepatic contribution, this score should be interpreted with caution. We did not assign a subgroup. Under the subclassification tree of these criteria, the absence of cutaneous manifestations would place the patient in the polymyositis group, whereas anti-MDA5 antibodies are associated with DM [1,2] and anti-ARS antibodies with the antisynthetase syndrome [2]. Because the classification tree and the serologic findings pointed to different subgroups, and because the antibodies were detected by ELISA alone without muscle histology, we use the broader term inflammatory myopathy in this report.

Our patient was positive for antibodies against both MDA5 and ARS. Coexistence of these antibodies is uncommon; in a cohort of 1280 patients with idiopathic inflammatory myopathies in which these antibodies were tested by immunoblotting and also by ELISA, only 6 (0.47%) were positive for both, and their clinical and radiologic features combined those associated with each antibody [7]. In contrast, the antibodies in our patient were detected by ELISA alone, without confirmation by a second method. In case reports, the clinical course has varied, from rapidly progressive and relapsing ILD in a patient with anti-MDA5 and anti-PL-7 antibodies [8] to a milder course in a patient with anti-MDA5 and anti-PL-12 antibodies [9]. Both of these patients had cutaneous involvement, whereas no rash developed in our patient at any point. On the basis of the available testing, the clinical features cannot be attributed to either antibody, and the findings discussed below, namely a normal CK, an elevated aldolase, hyperferritinemia, and a predominance of myalgia, do not depend on that attribution.

Requiring an elevated CK before considering an inflammatory myopathy would miss patients with inflammatory myopathies, including DM, who have a normal CK. In the series in which anti-MDA5 antibodies were first described, the CK was normal in 6 of 8 antibody-positive patients [1]. In a nationwide cohort of 121 anti-MDA5-positive patients, only 41.4%

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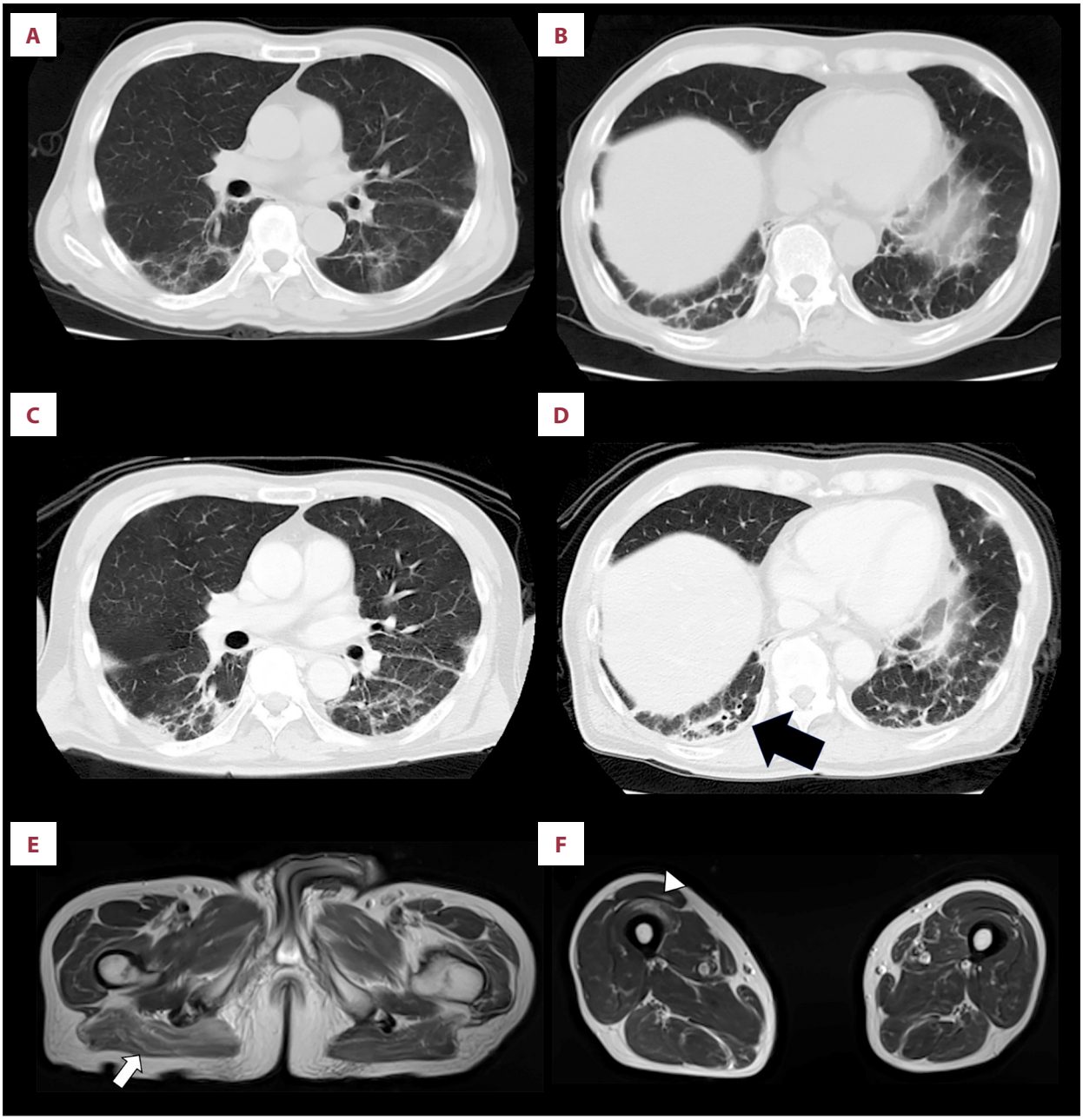


Figure 1. (**A, B**) Computed tomography scan of the chest, obtained on admission (Day 15), shows ground-glass opacities predominantly in the dorsal portions of both lower lobes. (**C, D**) Computed tomography scan of the chest, obtained on Day 19, shows progression of the ground-glass opacities to consolidation, with traction bronchiectasis in part (arrow). Panels **A** to **D** are conventional computed tomography images; thin-section reconstructions were not available. (**E, F**) Axial T2-weighted magnetic resonance imaging, obtained on Day 22 without fat suppression. (**E**) At the pelvic level, high signal intensity is seen in the gluteus maximus (white arrow). (**F**) At the mid-thigh level, high signal intensity is seen in the vastus intermedius (arrowhead), with a right-sided predominance.

of those evaluated had CK elevation [3], and 26% of patients with DM in a single-center series had a normal CK at presentation, with an elevated aldolase in most of those in whom it was measured [10].

In DM, the absence of a rash may be related to a normal CK. In a series of 34 patients with pathologically confirmed myopathy, an elevated aldolase, and a normal CK, DM was the most common diagnosis, accounting for 8 patients (23.5%). When these 8 patients were compared with 24 patients who had DM

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with an elevated CK, a skin rash was significantly less frequent in the normal CK group (50.0% vs 100.0%; $P = 0.002$) [11]. A normal CK and the absence of a rash thus appear to be associated rather than independent findings.

In our patient, the elevated aldolase with a persistently normal CK prompted evaluation for an inflammatory myopathy. Interpretation of aldolase requires caution. Transaminases and GGT were also elevated, raising the possibility of a hepatic source; Soontrapa et al excluded patients with elevated liver enzymes from their cohort for this reason [11]. T2-weighted MRI of the pelvis and thighs showed high signal intensity suggestive of muscle involvement. Aldolase is best regarded not as proof of muscle involvement but as an indication for further evaluation when the CK is normal.

Serum ferritin rose in parallel with the radiologic progression of ILD. In DM, ferritin predicts the development and severity of acute interstitial pneumonia and correlates with the prognosis [12]. Ferritin was markedly higher in patients with acute or subacute interstitial pneumonia than in those with chronic interstitial pneumonia or without ILD, and the 6-month survival rate was 28.6% when ferritin was 1500 ng/mL or higher, as compared with 82.5% when it was lower [12]. In our patient, ferritin was 1075 ng/mL on admission and rose to 3231 ng/mL within 3 days, while he was receiving prednisolone at a dose of 1 mg/kg/day, during which the ground-glass opacities progressed to consolidation. In our patient, hyperferritinemia may thus have reflected disease activity and rapid progression, although ferritin is not specific for DM and its predictive value cannot be established from a single case.

Our patient had predominant myalgia with a normal CK and no rash, and the diagnosis was difficult to establish. An initial suspicion of PMR has also been reported in a patient with anti-MDA5 antibody-positive ILD [13]. In both patients, glucocorticoids improved the musculoskeletal symptoms while the lung disease progressed. In that patient, the shoulder and wrist pain improved with prednisolone at 15 mg/day, and ILD was recognized after dyspnea developed; in our patient, the myalgia improved transiently with low-dose prednisolone and resolved with 1 mg/kg/day, while the ground-glass opacities progressed to consolidation. The CK was also normal and ferritin was elevated in that patient, but a V-sign and shawl sign were present and rheumatoid factor was positive. In our patient, no rash appeared at any point, and the incomplete response to low-dose prednisolone prompted the evaluation that identified the ILD before any respiratory symptoms had developed, as well as the muscle involvement suggested by aldolase elevation and MRI findings. The contribution of this report is the documentation of a rash-negative, normal-CK inflammatory myopathy that initially met a PMR classification score and later developed progressive ILD; the dual-antibody result forms part of this unusual context rather than a determinant of the phenotype. Even when myalgia predominates and PMR is suspected, an inflammatory myopathy should be included in the differential diagnosis if the response to low-dose glucocorticoids is inadequate.

This case report has limitations. First, muscle biopsy was not performed because of the progressive respiratory disease; classification therefore relied on the 2017 EULAR/ACR criteria without biopsy, and a subgroup could not be assigned [6]. Second, the 2 antibodies were detected by ELISA alone; confirmatory testing by immunoprecipitation was not performed, and the individual ARS specificity was not identified. Third, MRI was obtained without fat suppression, so the signal changes were less specific for muscle edema; together with the possible hepatic contribution to the aldolase level, the evidence of muscle involvement is supportive rather than definitive.

Conclusions

In conclusion, we report a case of clinically diagnosed inflammatory myopathy that mimicked PMR, presenting with predominant myalgia and without either a rash or CK elevation. Inflammatory myopathy should not be ruled out on the basis of a normal CK when a PMR-like presentation responds inadequately to low-dose glucocorticoids, and the differential diagnosis should be broadened. In this setting, an elevated aldolase level is a reason to investigate possible muscle involvement, and myositis-specific autoantibodies provide supportive evidence that must be interpreted together with the clinical phenotype and the testing method. In our patient, ferritin rose alongside radiologic progression of ILD and may have reflected disease activity in this clinical context; whether it predicts ILD more generally cannot be determined from a single case. Improvement of musculoskeletal symptoms with glucocorticoids does not exclude progression of lung disease. These findings may prompt earlier recognition and more timely evaluation of associated ILD.

Institution Where Work Was Done

Ito Municipal Hospital, Shizuoka, Japan.

Patient Consent Declaration

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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