ORIGINAL RESEARCH

DOI: 10.17944/interdiscip.1699543
Interdiscip Med J 2025;16(55):109-115

The clinical features and therapeutic results in patients diagnosed with idiopathic inflammatory myositis: single center experience

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Abstract

Objective: This study assessed clinical features, laboratory profiles, and treatment outcomes in idiopathic inflammatory myopathies (IIMs) patients after six months of immunosuppression.

Method: Retrospective analysis occurred at a tertiary rheumatology center. Baseline and six-month post-therapy assessments were conducted on patients diagnosed with polymyositis or dermatomyositis. Records were kept of clinical symptoms, laboratory markers, and muscle strength scores.

Results: With a mean age of 43±11.1 years and an almost even gender distribution (48.4% female), 31 patients were enrolled. Of these individuals, 87.1% were diagnosed with polymyositis and 12.9% with dermatomyositis. At baseline, the predominant symptom was muscle weakness (77.4%), alongside notably high levels of muscle enzymes (median creatinine kinase [CK]: 3340 IU/L) and inflammatory markers (C-reactive protein [CRP]: 11.4 mg/L, erythrocyte sedimentation rate [ESR]: 39 mm/h). Positivity for Jo-1 and Ro52 autoantibodies was found in 16.1% of the cases. Following six months of treatment, there were significant reductions in all laboratory markers ($p<0.001$), and all patients exhibited improved muscle strength (5/5).

Conclusion: Patients with IIMs experience significant biochemical and clinical improvement with immunosuppressive therapy. Optimal recovery and long-term muscle function depend on early diagnosis and personalized treatments.

Keywords: Dermatomyositis, idiopathic inflammatory myopathies, immunosuppressive therapy

INTRODUCTION

Idiopathic inflammatory myopathies (IIMs), a diverse group of autoimmune diseases, include polymyositis and dermatomyositis , which are marked by chronic muscle inflammation causing weakness and various extra-muscular manifestations (1). Both disorders present clinically with muscle weakness as a primary symptom. Dermatomyositis is known for unique skin symptoms like heliotrope rash, Gottron's papules, and photosensitivity, whereas polymyositis is mainly identified by muscle weakness without skin alterations (2,3). The shared pathophysiology of these conditions, specifically immune-mediated muscle fiber damage causing the observed symptoms, explains their classification as IIMs (4,5).

A wide array of autoantibody profiles within the IIM spectrum significantly influences the clinical expression, therapeutic response, and ultimate prognosis. Specifically, antibodies like anti-nuclear antibody (ANA), Jo-1, and Ro-52 are key here (6). Autoimmune diseases frequently show ANA presence, whereas the presence of Jo-1 antibodies, specific to anti-synthetase syndrome, is common in polymyositis and associated with a higher chance of interstitial lung disease (ILD) (7, 8). A milder myositis phenotype is often characterized by the presence of Ro-52 autoantibody, frequently alongside others, influencing patient outcomes (9,10).

Moreover, treatment plans are affected by the types of autoantibodies present, because certain autoantibodies

Cite this article: . Albayrak F, Babayiğit A, Çabuk Çelik N, Şahin A. The clinical features and therapeutic results in patients diagnosed with idiopathic inflammatory myositis: single center experience. Interdiscip Med J. 2025;16(55):109-115. https://doi.org/10.17944/interdiscip.1699543

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Received: May 14, 2025
Accepted: Aug 4, 2025

are associated with specific disease categories and the success of various treatments. Alternatively, patients exhibiting Jo-1 antibodies might respond better to different immunosuppressants than patients with Ro-52 positivity (7, 9). These findings underscore the value of a personalized therapeutic approach guided by autoantibody profiles. The significance of these profiles' predictive implications for disease cannot be overemphasized; autoantibodies like Jo-1 are linked to higher ILD rates, impacting survival and progression (2,3,5). Moreover, the clinical presentations associated with these autoantibodies often demonstrate a diverse array of non-muscle manifestations, which may result in complications impacting patients' quality of life (11). The diverse range of autoantibodies necessitates individualized approaches, since particular antibodies can forecast treatment response and long-term effects (12).

Given the intricate clinical and autoantibody characteristics of IIMs, the present study aimed to analyze the effects of these biochemical markers on disease characterization, treatment response, and prognosis. This study specifically sought to evaluate the clinical profiles, laboratory parameters, and therapeutic responses in patients diagnosed with IIMs. The effectiveness of standard immunosuppressive therapies (corticosteroids and other agents) on muscle enzyme levels, inflammatory markers, and muscle strength will be assessed by comparing data from baseline and six months after treatment.

METHOD

This retrospective observational study comprised 31 patients with diagnoses of IIM, polymyositis or dermatomyositis from a tertiary rheumatology center. Diagnostic criteria included established clinical features, muscle biopsy data, elevated muscle enzyme levels, and autoantibody profiles (1). Ethical approval was obtained prior to data collection, and all procedures complied with the principles of the Declaration of Helsinki. IIM patient data from Sivas Cumhuriyet University Faculty of Medicine were collected between 2021 and 2024. Sivas Cumhuriyet University Non-Interventional Clinical Research Ethics Committee approved the study (Decision protocol no: 2024/09-36).

To participate, individuals needed a confirmed polymyositis or dermatomyositis diagnosis, to be over 18, and to have completed at least 6 months of follow-up with accessible laboratory and clinical evaluations. Patients lacking complete records or those unavailable for follow-up after six months were omitted.

Electronic medical records were the source of demographic, clinical, and laboratory data. Collected data point encompassed age, sex, myositis type, co-morbidities, presenting symptoms, biopsy results, the presence of autoantibodies (Jo-1, Ro52), and baseline muscle strength. Muscle strength was measured with the Medical Research Council (MRC) scale; 0 represents no movement and 5 represents normal strength (13).

Alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatine kinase (CK), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and lactate dehydrogenase (LDH) serum levels were measured at the initial visit and after 6 months.

The primary treatment given to all patients was corticosteroid therapy. Treatment began with a personalized dose, was adjusted based on the patient's condition and progress. Disease severity, organ involvement, and treatment response guided the prescription of further immunosuppressants. The immunosuppressant treatment options included plaquenil (PLQ), mycophenolate mofetil (MMF), rituximab, cyclosporine, and intravenous immunoglobulin (IVIG), either as monotherapy or in combination.

The duration of steroid therapy and the most recent doses were recorded.

Statistical Analysis

Data management, visualization, and reporting were facilitated by various packages within R version 4.4.2 for the statistical analysis. Employing the R6 package yielded reusable object-oriented structures, thereby fostering more flexible and modular statistical workflows. The rstatix package simplified statistical tests significantly, while flextable was utilized to format analysis tables for publication-ready abstracts and journals.

Demographic and clinical characteristics were summarized using descriptive statistics. Continuous data were summarized as mean ± standard deviation (SD) if normally distributed, and as median (minimum–maximum) if non-normally distributed data. Frequencies and percentages showed the categorical variables.

Relationships and group differences were analyzed using inferential statistics. The normality of numerical data (Shapiro-Wilk test) and the satisfaction of test assumptions determined test selection. T-tests analyzed normally distributed numerical data from two independent groups, while ANOVA handled more than two groups. For non-normally distributed data, the Wilcoxon rank-sum test (two groups) or Kruskal-Wallis test (multiple groups) was applied. Categorical data analysis used Chi-square tests for sufficient cell observations (over 5), and Fisher's exact test for smaller samples. Treatment effect consistency was assessed within the larger PM patient subgroup via subgroup analysis. Results with $p < 0.05$ were deemed statistically significant.

RESULTS

This study included 31 IIM patients; 27 (87.1%) had polymyositis, and 4 (12.9%) had dermatomyositis. A mean age of 43±11.1 years was observed in participants, showing an almost even distribution of sex (48.4% female, 51.6% male). The mean time until diagnosis was 6.3±1.5 years. The main symptom reported was muscle weakness, affecting 77.4% of patients, while 22.6% experienced dysphagia. Osteoporosis (54.8%) and ILD (12.9%) were comorbid conditions. The demographic, clinical, and laboratory characteristics of myositis patients are displayed in Table 1.

Baseline laboratory parameters showed higher-than-normal levels, suggesting active myositis. The median serum AST was 88 U/L (66–110), ALT was 88 U/L (64–103), LDH was 478 U/L (389–589), and CK was significantly elevated at a median of 3340 IU/L (2100–4400). Elevated inflammatory markers included CRP at 11.4 mg/L (8.2–13.4) and ESR at 39 mm/h (36–43). Baseline muscle strength measured 3/5 (2–3).

Jo-1 and Ro52 autoantibodies were positive in 16.1% of patients. Myositis was confirmed by muscle biopsy in 41.9% (n=13) of patients, ruled out in 6.5% (n=2), and not tested in 51.6% (n=16).

Corticosteroids, at a median starting dose of 64.0 mg/day (48-80), formed the core of treatment regimens, with a median duration of usage of 28 months (23.5–36). At the final assessment, corticosteroid use was reported by just 16.1% of patients. The final median steroid dose was 4 mg/day for 64.5% and 2 mg/day for 19.4% of participants. Immunosuppressive agents given included plaquanil (58.1%), MMF (38.7%), IVIG (32.3%), rituximab (25.8%), and cyclosporine (9.6%). Some patients were treated with a combination of therapies.

Table 2 compares participants' laboratory results from their first visit and after six months of treatments. Six-month follow-up data revealed significant improvements in all clinical and laboratory measures. AST, ALT, LDH, and CK levels significantly decreased ($p<0.001$), from 88 to 24 U/L, 88 to 29 U/L, 478 to 156 U/L, and 3340 to 178 IU/L, respectively. Likewise, CRP declined from 11.4 to 4 mg/L ($p<0.001$), and ESR dropped from 39 to 20 mm/h ($p<0.001$). Marked muscle function recovery is indicated by the significant improvement ($p<0.001$) in muscle strength to 5/5 across all patients.

The polymyositis subgroup (n=27) showed similar trends to the overall study population (Table 3). Baseline levels for AST, ALT, LDH, and CK were 95 U/L (66–122), 98 U/L (64–104), 455 U/L (387–650), and 3340 IU/L (1496–4400), respectively. At baseline in polymyositis patients, inflammatory markers were elevated (CRP: 11.4 mg/L, ESR: 38 mm/h), and muscle strength was at 3/5.

Significant improvements were observed at 6 months, with AST, ALT, LDH, and CK levels dropping to 24 U/L, 28 U/L, 156 U/L, and 178 IU/L, respectively (all $p<0.001$). Muscle strength reached full recovery (5/5, $p<0.001$) as CRP and ESR levels significantly dropped ($p<0.001$) to 4 mg/L and 20 mm/h, respectively.

Table 1. Demographic, clinical and laboratory characteristics of participants diagnosed with myositis

Variables	**Overall**, n=31 n (%)	Variables	**Overall**, n=31 n (%)
Age*	43±11.1	Baseline LDH (U/L)*	478 (399.5-583.5)
Sex (Female/Male)	15 (48.4)/16 (51.6)	Baseline CK (IU/L)*	3340 (2115-4360)
Type of myositis Polymyositis Dermatomyositis	 27 (87.1) 4 (12.9)	Baseline CRP (mg/L)*	11.4 (8.6-13.0)
Average time to diagnosis*	6.3±1.5	Baseline ESR (h)*	39 (36-43)
Complaint at the time of admission Muscle weakness Difficulty in swallowing	 24 (77.4) 7 (22.6)	Baseline muscle strength	3/5 (2-3)
Biopsy result Positive Negative Not done	 13 (41.9) 2 (6.5) 16 (51.6)	Jo1 positivity	5 (16.1)
Osteoporosis	17 (54.8)	Ro52 positivity	5 (16.1)
ILD	4 (12.9)	Follow AST (U/L)*	24 (22-26)
Steroid starting dosage (mg/days)	64 (48-80)	Follow ALT (U/L)*	29 (24.5-31)
Minimum steroid usage duration (months)	28 (23.5-36)	Follow LDH (U/L)*	156 (144-171.5)
Steroid continuation status Yes No	 5 (16.1) 26 (83.9)	Follow CK (IU/L)*	178 (170-188)
Last steroid dosage 2 (mg/days) 4 (mg/days)	 6 (19.4) 20 (64.5)	Follow CRP (mg/L)*	2 (1-2.5)
Immunosuppressive treatments** Plaquenil MMF IVIG Rituximab Cyclosporine	 18 (58.1) 12 (38.7) 10 (32.3) 8 (25.8) 3 (9.6)	Follow ESR (h)*	20 (19-21.5)
Baseline AST (U/L)*	88 (66.5-105.5)	Follow muscle strength	5/5 (5-5)
Baseline ALT (U/L)*	88 (66-102)		

*Numeric variables were presented as median (minimum–maximum) or mean ± SD. **Some patients received combined therapy. **Abbreviations**: ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CK: Creatine kinase; CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate; ILD: Interstitial lung disease; IVIG: Intravenous immunoglobulin, h: hours; LDH: Lactate dehydrogenase; L: Litre; mg: milligram; MMF: Mycophenolate mofetil; U/L: Unit/Litre.

DISCUSSION

This study presents a complete examination of clinical traits, laboratory parameters, and therapeutic outcomes for a group of patients with IIMs, with a predominant subset diagnosed with polymyositis. Immunosuppressive therapy

significantly improved the clinical and biochemical status of IIM patients in the current study. Lower muscle enzymes, less inflammation, and increased strength in myositis patients demonstrate the treatment's effectiveness. Within six months of treatment, a significant number of patients achieved remission or substantial disease control. The study's results expand knowledge on IIMs, emphasizing immunomodulatory treatments' benefits.

Table 2. Comparisons of laboratory measurements of all participants at the first visit and at 6 th months of treatment

	At first visit, n=31	6 th months of treatment, n=31	p value
AST (U/L)*	88 (66-110)	24 (22-26)	<0.001
ALT (U/L)*	88 (64-103)	29 (24-31)	<0.001
LDH (U/L)*	478 (389-589)	156 (144-172)	<0.001
CK (IU/L)*	3340 (2100-4400)	178 (170-188)	<0.001
CRP (mg/L)*	11.4 (8.2-13.4)	4 (3-5)	<0.001
ESR (h)*	39 (36-43)	20 (19-22)	<0.001
Muscle Strength	3/5 (2-3)	5/5 (5-5)	<0.001

*Numeric variables were presented as median (minimum–maximum). Abbreviations: ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CK: Creatine kinase;CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate; L: liter, mg: milligram, U/L: Unit/Litre.

Table 3. Comparison of laboratory measurements of patients diagnosed with polymyositis at the first visit and at 6 th months of treatment

	At first visit, n=31	6 th months of treatment, n=31	p value
AST (U/L)*	95 (66-122)	24 (22-26)	<0.001
ALT (U/L)*	98 (64-104)	28 (24-30)	<0.001
LDH (U/L)*	455 (387-650)	156 (144-171)	<0.001
CK (IU/L)*	3340 (1496-4400)	178 (170-188)	<0.001
CRP (mg/L)*	11.4 (8-13.7)	4 (3-5)	<0.001
ESR (h)*	38 (34-43)	20 (19-22)	<0.001
Muscle strength	3/5 (2-3)	5/5 (5-5)	<0.001

*Numeric variables were presented as median (minimum–maximum). **Abbreviations:** ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CK: Creatine kinase; CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate; L: litre; mg: milligram; U/L: Unit/Litre.

The characteristics of the study group, with a mean age of 43 years and a nearly equal gender distribution, are similar to previous IIM studies (1, 5), which shows that the condition most often affects adults aged 30–60, with no clear preference for one sex in polymyositis, unlike dermatomyositis, which tends to affect female more. The roughly 6.3-year average time from symptom onset to diagnosis in autoimmune myopathies likely reflects the often-reported diagnostic delays due to the diseases' insidious onset and non-specific symptoms (14).

Consistent with the cardinal clinical hallmark of myositis, which is typical symmetric, proximal muscle weakness (15), most patients (77.4%) presented with muscle weakness in the present study. Dysphagia, affecting 22.6% of patients, is a known symptom of polymyositis, stemming from the involvement of pharyngeal and upper esophageal skeletal muscles (16).

Baseline laboratory parameters showed higher-than-normal levels, indicative of active disease process: elevated serum CK, LDH, and liver transaminases (AST, ALT), which can also be released from damaged skeletal muscle (17). These elevations are useful as both diagnostic markers and indicators of disease activity (18). Levels of CK, with a median of 3340 IU/L, are comparable to those in cohorts exhibiting active polymyositis and dermatomyositis (19).

Elevated inflammatory markers, CRP and ESR, confirm the systemic inflammation in the disease. Even though hepatic regulation and cytokine profiles (e.g., IL-6) often keep CRP within normal ranges during IIM, the current study data align with findings of elevated CRP in more active or systemic disease (20).

Autoantibody profiling revealed that 16.1% of patients tested positive for anti-Jo-1, an antisynthetase antibody that is strongly associated with polymyositis and ILD, which was present in 12.9% of this cohort. Anti-Ro52 antibodies, present in 16.1% of patients, frequently occur alongside anti-Jo1 antibodies, correlating with worsened disease severity and ILD (2,5). Patient stratification for pulmonary involvement risk and tailored immunosuppression rely heavily on these autoantibodies (21).

Muscle biopsies confirmed the diagnosis in 41.9% of cases; however, 51.6% did not undergo the procedure, potentially due to sufficient non-invasive data or the invasiveness of the procedure. However, if there is uncertainty, histopathology remains the top diagnostic method (1,22).

A median starting dose of 64.0 mg/day of glucocorticoids was given to all patients as a first-line treatment for a median duration of 28 months. With a typical dose of steroid treatment(0.5–1 mg/kg/day), the present study group showed a significant clinical response, hinting at ethnic or disease severity-related variations in glucocorticoid sensitivity (23).

Plaquenil (58.1%), MMF (38.7%), IVIG (32.3%), rituximab (25.8%), and cyclosporine (9.6%) were the immunosuppressive therapies administered. With mounting evidence (1,16, 24), these agents are proving to be effective steroid-sparing treatments in refractory or steroid-intolerant cases. Notably, Rituximab shows promise against anti-synthetase syndrome and Jo1-positive myositis, as indicated by its use in Jo1-positive patient group (25).

Statistically significant improvements in all biochemical and clinical parameters were observed following a six-month treatment period. AST, ALT, LDH, and CK levels showed a significant decrease ($p<0.001$ for all), correlating with clinical

remission and resolution of muscle injury. The marked reduction in muscle enzyme and inflammatory markers between baseline and follow-up assessments reliably demonstrates the effectiveness of treatment and successful disease management in patients with IIM, particularly polymyositis. Monitoring these biochemical changes is key to evaluating the success of therapeutic interventions. Reduced muscle enzyme levels generally indicate decreased muscle injury and inflammation, consistent with treatment outcomes (1,26). Reductions in CRP and ESR indicated effective control of systemic inflammation. This finding reinforces the idea that anti-inflammatory treatments improve muscle problems and reduce overall immune system activation in myositis (27). In addition, the significant drop in muscle and inflammation markers could also indicate how well immunosuppressive treatments work in reducing the autoimmune response in polymyositis. The normalization of enzyme levels is anticipated as the inflammatory process resolves with ongoing treatment. In this context, the link between lower muscle enzyme levels and better patient outcomes supports the idea that blood markers are useful for diagnosis, treatment assessment, and disease progression (26,27,28). Moreover, interpreting these results indicates that routine biochemical assessments in a structured follow-up program would improve patient management. Identifying inadequate treatment responses early enables timely therapeutic changes, increasing chances of disease control (15,29). Therefore, incorporating routine laboratory monitoring into care for IIM improves individualized treatment and results.

The greatest improvement was in muscle strength, increasing from 3/5 to 5/5 ($p<0.001$), representing near-total functional recovery. The progress achieved emphasizes the possibility of fully restoring muscle function through timely, effective treatment, corroborating findings from controlled clinical trials and real-world cohorts (5).

The effectiveness of immunosuppressive treatments in this patient group is further supported by the fact that the polymyositis subgroup (87.1%), regardless of diagnosis, experienced substantial biochemical and functional gains. Notably, improvements across all parameters in this subgroup support the conclusion that these benefits are not limited to specific myositis types but generalize across the idiopathic spectrum.

The high prevalence of osteoporosis (54.8%) in patients necessitates a focus on proactive bone health monitoring and preventive measures due to the long-term steroid use in chronic myositis (30). Six months later, the encouraging results notwithstanding, steroid use remained low at 16.1%. This might point to effective disease control, but it could also be because doctors prefer to decrease treatment due to side effects, instead of sustained remission. Future studies would benefit from a detailed analysis of relapse rates after steroid tapering.

Limitations of the Study

The systematic evaluation of a well-defined cohort, with comprehensive follow-up and assessment of objective biomarkers and functional outcomes, is the key strength of the current study. However, this study is limited by its small sample size, the lack of a comparison group, and potential referral bias from its single-center design. In addition, including both polymyositis and dermatomyositis, though polymyositis is more prevalent, might cause variability in phenotype. Further research needs to validate these findings in larger, multi-center studies with extended follow-up to better characterize relapse rates, remission durability, and long-term functional status. Additionally, incorporating newer biomarkers such as anti-melanoma differentiation-associated gene5 , transcription intermediary factor 1 gamma, and cytokine panels might enhance our understanding of pathophysiologic mechanisms and treatment response heterogeneity (28).

CONCLUSION

This research confirms that immunosuppressants significantly improve IIM, especially polymyositis, biochemical markers, and muscle function within six months. Optimal patient outcomes necessitate early diagnosis, rigorous laboratory monitoring, and personalized immunomodulatory approaches. The notable decline in muscle enzymes and inflammatory markers highlights the success of current treatments. Further multicenter studies should validate these findings, assess relapse rates, and improve long-term myositis disease management.

ACKNOWLEDGEMENTS

Peer-Review: Both externally and internally peer reviewed.

Conflict of Interest: The authors declare that they have no conflict of interests regarding content of this article.

Financial Support: The authors report that this work was self-funded and that no additional grants were received.

Ethical Declaration: Ethical approval was obtained from Sivas Cumhuriyet University Non-Interventional Clinical Research Ethical Committee (Decision protocol no: 2024/09-36), and Helsinki Declaration rules were followed to conduct this study.

Authorship Contribution: Concept: FA, AB, NÇÇ, AŞ, Design: FA, AB, Supervising: FA, AŞ, Financing and equipment: FA, AB; NÇÇ, AŞ, Data collection and entry: FA, AB, NÇÇ, Analysis and interpretation: FA,, Literature search: FA, AB, NÇÇ, AŞ, Writing: FA, AB, NÇÇ, AŞ, Critical review: FA, AŞ:

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